Literature Review

A Systematic Review of Sleep and Circadian Rhythms in Children with Attention Deficit Hyperactivity Disorder

Journal of Attention Disorders
2022, Vol. 26(2) 149–224

DOI: 10.1177/1087054720978556
journals.sagepub.com/home/jad
SAGE

Upasana Bondopadhyay[1], Unai Diaz-Orueta[1], and Andrew N. Coogan[1]

Abstract

Objective: Children and adults with ADHD often report sleep disturbances that may form part of the etiology and/or symptomatology of ADHD. We review the evidence for sleep changes in children with ADHD. **Methods:** Systematic review with narrative synthesis assessing sleep and circadian function in children aged 5 to 13 years old with a diagnosis of ADHD. **Results:** 148 studies were included for review, incorporating data from 42,353 children. We found that sleep disturbances in ADHD are common and that they may worsen behavioral outcomes; moreover, sleep interventions may improve ADHD symptoms, and pharmacotherapy for ADHD may impact sleep. **Conclusion:** Sleep disturbance may represent a clinically important feature of ADHD in children, which might be therapeutically targeted in a useful way. There are a number of important gaps in the literature. We set out a manifesto for future research in the area of sleep, circadian rhythms, and ADHD.

Keywords

ADHD, children, sleep, circadian: systematic, actigraphy, circadian rhythms

Introduction

Attention Deficit Hyperactivity Disorder (ADHD) is a common neurodevelopmental disorder characterized by the core psychopathological features of inattention, hyperactivity and impulsivity, and is associated with poor psychosocial outcomes (McGough, 2014). Children diagnosed with ADHD exhibit goal directed behaviors, although these frequently are incomplete or inaccurate, resulting in impaired outcomes for academic, vocational and interpersonal interactions, accompanied by maladaptive psychological and physiological traits (Posner et al., 2020). ADHD, like other neurodevelopmental disorders, has been associated with sleep disturbances which may manifest as long sleep latency, sleep phase delay syndrome, increased periodic limb movements during sleep, daytime sleepiness, altered total sleep duration and difficulty initiating and maintaining sleep (Ball, 1997; Corkum et al., 1998; Cortese et al., 2006; Konofal et al., 2001, 2010; Mayes et al., 2009). As sleep deprivation may exert multiple impacts on neurobehavioral and cognitive systems, including attention and emotional regulation (Krause et al., 2017), sleep disturbances in ADHD may affect the core psychopathology of the condition, and as such sleep-related factors may be important in the etiology of the condition. Further, sleep disturbances may arise from ADHD-related impulsivity and hyperactivity, and as such sleep problems may be part of the symptomatology of ADHD (Raman and Coogan, 2019).

Sleep is a highly complex, dynamic process involving multiple stages (including rapid eye movement (REM) sleep and non-REM stages) and may serve a number of essential, non-redundant functions (Scammell et al., 2017). In order to understand how sleep and ADHD may be related, it is important to examine key behavioral control systems that shape sleep physiology and sleep behavior. The sleep/wake cycle is shaped by the interaction of the circadian clock and the sleep homeostat (Borbély et al., 2016). The circadian clock is an endogenous daily timekeeping mechanims that produces rhythms with periods of near 24 hours in a host of physiological processes (Vitaterna et al., 2001). Sleep homeostasis adjusts sleep processes to account for time spent in wakening and accumulated sleep pressure since the last consolidated sleep bout (Deboer, 2018). Dysfunction of the circadian system and/or the sleep homeostat may contribute to sleep problems in ADHD (Bijlenga et al., 2019). Studies of children, adolescents and adults with ADHD, utilizing both objective and subjective assessment of rest and activity cycles show significant variations in daily rhythms in behavioral, cognitive,

[1]Department of Psychology, Maynooth University, National University of Ireland, Maynooth

Corresponding Author:
Andrew N. Coogan, Department of Psychology, Maynooth University, National University of Ireland, Maynooth, Kildare, Ireland.
Email: andrew.coogan@mu.ie

endocrine, physiological and molecular processes, when compared to typically functioning individuals (Korman et al., 2019). Studies of circadian rhythms in adults with ADHD consistently report alterations in circadian phase, chronotype and other circadian parameters, indicating that the circadian clock may play an important role in adult ADHD (Coogan and McGowan, 2017).

As sleep and circadian function are profoundly influenced by life course (Roenneberg et al., 2007), there may be differential relationships of sleep, circadian rhythms and ADHD symptoms across the life course (Hegarty et al., 2019). This may be most marked during adolescence given the neurodevelopmental, behavioral and sleep-related changes that occur during this period (Pokhrel et al., 2013). As such, to gain a comprehensive insight into such relationships, it may be most useful to examine relatively narrow age ranges, especially in childhood and adolescence.

In the present review, we sought to synthesize the current evidence relating to sleep and circadian function in children between the ages of 5 and 13. We identified the need for such a review through a survey of the extant systematic reviews on sleep and/or circadian rhythms and ADHD via the Prospero and Cochrane registers of existing reviews in this field (supplementary material). The existing literature reviews in this topic either narrowly concentrate upon discussing possible treatment strategies and therapeutic approaches (Alamar et al., 2015; Bioulac et al., 2015; Cortese et al., 2013; Hvolby et al., 2015; Nikles et al., 2020; Tsai et al., 2016), or review the relationships between specific sleep features and ADHD in smaller systematic reviews focused on specific sleep feautres (Martin et al., 2019; Mogavero et al., 2018; Polmann et al., 2020; Sadeh et al., 2006; Martins et al., 2019; Scarpelli et al., 2019; Walters et al., 2008). Further, a number of reviews focus generally on the pharmacotherapy of ADHD, and as such sleep issues are of secondary interest in these works (Abdelgadir et al., 2018; Cortese et al., 2018; Krinzinger et al., 2019; Punja et al., 2016; Storebø et al., 2016, 2018). Although, a selected group of reviews do explore sleep and circadian rhythms in ADHD, they do so for adult ADHD (Coogan and McGowan, 2017; Lugo et al., 2020; Díaz-Román et al., 2018; Wajszilber et al., 2018). Given that more than half of the ADHD diagnosis is made during childhood (Pastor & Reuben, 2008; Visser et al., 2010) clearly defining the changes in sleep/wake rhythms in the childhood ADHD population is vital. As such, we have identified a need for an up-to-date, systematic and broad review of the association of sleep and ADHD in childhood, and it is to this need that the current study is addressed.

Methods

The method of this systematic review was developed in accordance to the Preferred Reporting Items for Systematic Reviews and Meta-Analysis (PRISMA) criteria (Liberati et al., 2009). Given the wide variety of sleep and circadian rhythms measurements used in extant studies reviewed, and the broad nature of the review, we decided that meta-analysis would not be useful given the high level of methodological heterogeneity. Acknowledging this methodological heterogeneity in the current literature, limiting our study to a meta-analysis would limit our discussion to only a specific assessment modality, thereby restricting the scope of our project. Rather, we sought to systematically review the literature and conduct a narrative synthesis.

Study Eligibility Criteria

For inclusion, studies must have had clearly identifiable cohorts of children between the age group of 5 to 13 years old with a diagnosis of ADHD through standardized diagnostic tools. We have restricted our participant criteria to ADHD diagnosed individuals because including a general population or clinical sample of children would lead to the overlapping presence of features characterizing different neurodevelopmental disorders, developmental delays and other psychiatric disorders. Any studies in humans that discussed findings of sleep or circadian functioning (subjectively or objectively measured through sleep logs, questionnaires, psychological tests, actigraphy, polysomnography or other physiological techniques) in cohorts with clinical assessment of ADHD symptoms were included for further reading, given that they adhered to the following: is an original, peer-reviewed and published paper that discussed empirical primary study findings (conference abstracts, book chapters, academic letters and reviews were excluded); the language of the article was English; and the publication date was between 1st January 2009 to 31st December 2019.

Search Strategy, Screening, and Extraction

We used the search logic ("Attention Deficit Hyperactivity Disorder" OR "ADHD" OR "Hyperkinetic disorder") AND ("Sleep" OR "Circadian" OR "Chronotype" OR "Actigraphy" OR "Polysomnography" OR "Diurnal Preference") to search PubMed, Embase, Web of Science and Clinical trials.org databases for studies published between 1st January 2009 and 31st December 2019. After recording search results from individual databases, all results were consolidated in the EndNote application and the first attempt at removing the duplicates from each database search was made. The resultant record of 2,540 studies were then uploaded to the Rayyan website (https://rayyan.qcri.org/), an online application for creating article databases for systematic reviews. From this initial group of studies, all study abstracts were screened to be filtered (based on previously decided inclusion criteria) in to "included", "excluded", "maybe" and duplicate categories. The "maybe" category was subsequently reviewed by all

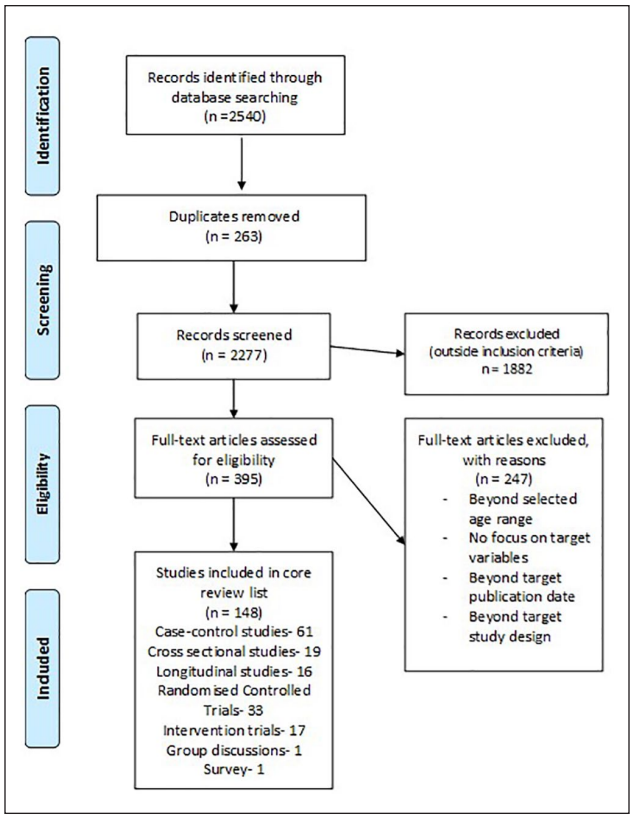

Figure 1. PRISMA flowchart representing the step by step exclusion leading to the final group of selected studies.

three authors to reach a consensus about being either included in the core review list of studied articles or completely excluded. Next, the "included" list of studies were further discussed in detail to filter out articles that were not congruent with the inclusion criteria.

Figure 1 includes the PRISMA flowchart representing the step by step exclusion leading to the final group of selected articles. The process of data extraction included the first author selectively mining the following information from the articles to form a master excel sheet of studies included in the core review list: study title; authors; study design; study objective; participant characteristics (including sample size, diagnosis, gender, ADHD medication use, and age); primary measures used; limitations and summary of main results.

Results

Overall 148 articles were included for review, encompassing findings from a total 12427 children diagnosed with ADHD, 29,068 typically developing children and 858 children with other clinical/psychiatric conditions. The selected pool of articles consisted of 61 case control studies, 19 cross sectional studies and 16 longitudinal studies. For the psychological and pharmacotherapy-based intervention studies, 33 randomized controlled trials, 17 intervention trials, and 1 each of group discussion and survey method study were recorded. For structured review of these studies, we categorized them into five sections: (1) articles exploring sleep in children diagnosed with ADHD; (2) articles exploring circadian rhythms and chronotype in children diagnosed with ADHD; (3) articles exploring functional consequences of sleep functioning in children with ADHD; (4) articles exploring sleep-related interventions and their impact on sleep and daytime function in childhood ADHD, and (5) articles exploring childhood ADHD pharmacotherapy effects on sleep. Studies from each category has been represented in separate tables (Supporting material B).

Sleep in Children Diagnosed with Attention Deficit Hyperactivity Disorder

Subjective assessment of sleep in children with ADHD through Parental/caregiver/teacher reports. In this section the studies reviewed report differences in parent-rated sleep variable measures for children with ADHD when compared to a typically functioning group (study details provided in Table 1). Significantly higher parental ratings on the Child Sleep Habits Questionnaire (CSHQ) for bedtime resistance, sleep anxiety, parasomnias, sleep onset delay, sleep disordered breathing, day time sleepiness and a global sleep disturbance score was found among children with ADHD when compared to age matched control group (Abou-Khadra et al., 2013; Chiraphadhanakul et al., 2015; Moreau et al., 2013). Exploring the above variables further, Lycett, Mensah, et al. (2015) found that a parent reported 7-day sleep log and CSHQ ratings revealed a distinct sleep pattern: children with moderate/severe sleep problems had shorter sleep duration (by −30 minutes), more night awakenings and longer sleep latency when compared to those with no/mild sleep problems. Additionally, Lycett, Mensah, et al. (2014) found that among 195 children with ADHD (5–13 years), 49% had transient parental-reported sleep problems (assessed through CSHQ), whereas 10% were reported to have persistent sleep problems. For persistent sleep problems, risk factors were found to be internalizing and externalizing symptoms severity, medication use and ADHD symptoms severity, whereas for transient sleep problems, poor parental mental health and co-occurring internalizing and externalizing behaviors were found to be risk factors (Lycett, Sciberras, et al., 2014). Using the CSHQ, it was also found that elevated scores on individual subscales (sleep onset latency, night waking, daytime sleepiness, sleep duration, parasomnias, daytime sleepiness) among the ADHD cohort were positively associated with a variety of parent reported externalizing behavioral problems (social, attentional and disruptive behaviors) and functional impairment (family, school, life skills, social activities and risky actions) (Aronen et al., 2014; Choi et al.,

Table 1. Summary of Thirty One Included Studies Focusing on Subjective Measures of Sleep in ADHD.

		Participant characteristics						Summary of main results
Authors/ publication year	Study design	Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age	Primary measures, limitations and biases	Objectives	Interventions/comparisons/ outcomes
Abou-Khadra et al. (2013)	Case control	103 (ADHD: 41, control: 62)	85.4%/14.6%	Not currently medicated.	6–12years	CSHQ, CPRS-R:L, Ferritin AccuBind ELISA test. Limitations: Small sample size, parent reports might lead to over-under estimation of sleep concerns and symptom severity, psychiatric clinic cases of ADHD might differ from community children with ADHD.	To examine the relationship between parent reported sleep problems, symptom-ratings, and low serum ferritin levels among Egyptian children with ADHD.	ADHD group showed significantly higher scores in CSHQ subscales (bedtime resistance 11.9 ± 3.0 vs. 9.4 ± 2.2, $p < .001$), sleep anxiety (8.0 ± 2.4 vs. 6.1 ± 2.0, $p < .001$), parasomnias (11.9 ± 2.9 vs. 9.6 ± 3.3, $p = .001$), sleep-disordered breathing (4.6 ± 1.9 vs. 3.9 ± 1.5, $p = .046$), daytime sleepiness (15.9 ± 3.5 vs. 14.2 ± 3.3, $p = .018$) and total scale score (60.0 ± 10.4 vs. 51.5 ± 9.2, $p < .001$) and negative association between low serum ferritin levels and sleep disturbances ($r = -.363$, $p = .020$).
Moreau et al. (2014)	Case control	ADHD: 41, control: 41	58.5%/41.5%	31 ADHD children on medication (Psychostimulants and Atomoxetine), 10 not currently medicated	6–13years	Actigraphy, Sleep Diary, CSHQ (Child Sleep Habits Questionnaire), BRIEF (Behavior Rating Inventory for Executive Functions), Conner's CPT (Continuous Performnace Test), CPRS (Conner's Parent's Ratings Scale), K-SADS (Kiddie-Schedule of Affective Disorders and Schizophrenia), Insomnia Severity Index for children (ISI-C). Limitations: Almost one-half of the sample was composed of children with predominantly inattentive subtype, rating scales used to define comorbidity might not be accurate, given the small sample size, only ten children were unmedicated.	To examine the sleep of children with ADHD, using actigraphy and parental questionnaires, and examine the potentially moderating role of psychostimulant medication and psychiatric comorbidity.	Children with ADHD significantly differed (with higher scores) from controls on (CSHQ total score 42.75 ± 6.27 vs. 37.49 ± 3.70, $p < .001$), sleep onset delay (1.68 ± .79 vs. 1.17 ± .50, $p < .01$), sleep anxiety (5.77 ± 1.66 vs. 4.55 ± 1.04, $p < .001$), parasomnias (8.80 ± 1.56 vs. 7.91 ± 1.04, $p < .01$), daytime sleepiness (11.08 ± 3.14 vs. 3.93 ± 2.61, $p < .05$), ISI-C total score (9.36 ± 5.66 vs. 3.01 ± 2.69, $p < .001$) and actigraphic measures of sleep (total sleep time (466.4 ± 40.6 vs. 490.6 ± 33.3, $p < .01$), sleep onset latency (32.7 ± 16.13 vs. 20.81 ± 10.07, $p < .001$), sleep efficiency (79.21 ± 4.99 vs. 82.03 ± 3.71, $p < .01$), mean activity counts per epoch (22.28 ± 10.15 vs. 14.11 ± 4.94, $p < .001$).
Chirapadhanakul et al. (2015)	Case control	ADHD: 55, control: 110	81.8%/18.2%	27 ADHD children on medication (short or long acting MHP)	5–12years	CSHQ (Thai version), the ADHD rating scales, and the Strengths and Difficulties Questionnaire (SDQ). Limitations: Sleep difficulties obtained by only parent report, marginal significance in father's education and income between both groups of study participants, and the lack of psychometric validation of the CSHQ–Thai version.	To compare the sleep disturbances in Thai children aged 5–12years with ADHD and typically developing children using the Children's Sleep Habits Questionnaire (CSHQ)–Thai version.	Children with ADHD had significantly higher scores in all subscales of the CSHQ (@ $p < .05$) and 58.2% of ADHD children with higher SDQ scores (>15) appeared to have more sleep disturbances (including bedtime resistance (mean ± SD = 12.55 ± 2.62 vs. 10.52 ± 2.63, $p = .005$), sleep anxiety (mean ± SD = 8.23 ± 2.06 vs. 6.30 ± 2.05, $p = .003$), parasomnias (mean ± SD = 10.41 ± 3.72 vs. 8.84 ± 1.53, $p = .036$), and total sleep disturbances (mean ± SD = 55.81 ± 7.18 vs. 49.14 ± 5.93, $p = .001$) on the CSHQ).

(continued)

Table 1. (continued)

Authors/ publication year	Study design	Participant characteristics				Primary measures, limitations and biases	Objectives	Summary of main results
		Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age			Interventions/comparisons/ outcomes
Lycett, Mensah, et al. (2015)	Cross sectional	ADHD: 392	85%/15%	313 ADHD children on medication	5–13 years	7-day sleep log, CSHQ, ADHD-RS-IV, sleep problems severity assessment, Depression anxiety stress scale, Anxiety Disorders Schedule for Children-IV, Socio-Economic Indexes for Areas. Limitations: All sleep measure's reliance on parental report reflect common effect of parental perception across the measures, findings limited to behavioral sleep problems, two-thirds of parents returned the sleep log, which may have under- or over-ascertained problem sleepers.	To compare parent report on a global measure of the severity in child sleep problem (no/mild vs. moderate/severe) with parent report on a 7-Day Sleep Log and the CSHQ.	Sleep log data identified 2 distinct sleep patterns according to parent-reported sleep problem severity (no/mild & moderate/severe). Children with moderate/severe sleep problems slept, on average, 30 minutes less per night; and were more likely to experience night awakenings and had more problematic scores (effect sizes: .5–1.1) across all domains of CSHQ in comparison to those with no/mild sleep problems.
Lycett, Mensah, et al. (2014)	Cross sectional	ADHD: 195	87.2%/12.8%	152 ADHD children on medication (Psychostimulants and Atomoxetine)	5–13 years	CSHQ, subjective sleep problems reported, Anxiety Interview Schedule for children/parent, ADHD rating scale-IV and Depression Anxiety Stress Scale for caregiver. Limitations: children in persistent sleep problems trajectory was small ($n=20$), leading to insufficient power to demonstrate relationships precisely, age range of children spanned 9 years; therefore study unable to examine age-specific sleep problem trajectories, sleep medication (e.g., melatonin) data were not available at all three time points; thus, it is difficult to determine whether use of sleep medication altered trajectories.	To examine behavioral sleep problem trajectories, types of sleep problems experienced, and associated risk/ protective factors among ADHD children.	Sleep problems in children with ADHD are commonly transient, but in a subgroup they are characterized as persistent. The most common trajectory was transient (49%; 95% CI 42, 56), followed by never (41%; 95% CI 34, 48) and persistent (10%; 95% CI 6, 15).
Choi et al. (2010)	Case control	ADHD: 27, control: 26	88.9%/11.1%	Medication naïve.	7–12 years	CSHQ, CBCL (Child Behavior Checklist), K-SADS-PL-K, PSG. Limitations: PSG results not controlled for first-night effect in children with ADHD who are more sensitive to changes in environment than controls, children got only 6 hours sleep on the night of the N-PSG compared to 9 hours at home, small sample, mainly boys, correlation coefficients were small and the relationship was modest.	To assess sleep characteristics in children with ADHD through polysomnographic recordings and parental reports of sleep problems.	Reported sleep problems were significantly associated with almost all subscales of CBCL as well as CBCL total score (between parasomnia and withdrawn $r=.286$, $p<.05$, between total sleep disturbance and withdrawn $r=.290$, $p<.05$, between somatic complaints and total sleep disturbance $r=.331$, $p<.05$, between anxious/depressed and total sleep disturbance $r=.366$, $p<.01$ between social problems and total sleep disturbance $r=.331$, $p<.01$ between attention problems and total sleep disturbance $r=.331$, $p<.01$, between aggressive behavior and total sleep disturbance $r=.376$, $p<.01$, between externalizing score and total sleep disturbance $r=.374$, $p<.01$, between CBCL total score and total sleep disturbance $r=.429$, $p<.01$.

(continued)

Table 1. (continued)

Authors/ publication year	Study design	Participant characteristics: Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age	Primary measures, limitations and biases	Objectives	Summary of main results: Interventions/comparisons/ outcomes
Eyuboglu and Eyuboglu (2018)	Case control	ADHD: 83, control: 106	77.1%/22.9%	Participants not currently medicated.	6–12 years	Conner's parent questionnaire, CSHQ, Weiss Functional Questionnaire. Limitations: ODD was not excluded, restless leg syndrome and periodic leg movement disorder were not evaluated, all scales were completed by parents; thus not controlling effect of parental perception on measures, family sleeping attitudes, daily physical activity, and use of electronics before bedtime were not examined.	To evaluate sleep problems of un-medicated children with ADHD and to investigate the effects of these problems in functionality.	Children with ADHD experienced more sleep problems and slept less than healthy children (total sleep disturbances mean ± SD = 56.3 ± 10.2 vs. 42.9 ± 6.1, $p < .001$ on CSHQ; amount of sleep (hours) mean ± SD = 8.5 ± 1 vs. 10.3 ± 1.2, $p < .001$.), and functional impairments increased due to these problems ($r = .707$, $p < .001$).
Aronen et al. (2014)	Case control	CD & ODD: 30 (16 comorbid ADHD), control: 30	90%/10%	11 children with ADHD on medication (Risperidone and MPH)	7–12 years	K-SADS, CSHQ, Actigraphy. Limitations: Small sample size, likely limiting the statistical power of the tests used, the actigraphic measurement time (3 nights) was only modest.	To compare the self- and parent-reported sleep problems and objectively measured sleep amount and efficiency in child patients with ADHD/ CD/ODD and their age- and gender-matched controls.	Children with comorbid ADHD slept significantly less than did the patients with CD/ODD alone and the controls (total sleep minutes mean ± SD = 500.3 ± 50.2 (ADHD+CD/ ODD) vs. 416.3 ± 59.8 (CD/ODD), $p < .001$ as measured through actigraphy).
Sciberras et al. (2016)	Case control	ADHD: 177, control: 212	68.93%/31.07%	21 children with ADHD on medication (MPH)	6–8 years	Subjective questions for sleep problems, Daignostic Interview schedule for children-IV, BMI z-score, Child health questionnaire. Limitations: Measures of sleep, physical injuries and global health were brief and reported solely by parents, unable to verify the type of sleep problem(s) experienced by children, number of children in our subgroup analyses examining differences by ADHD subtype was small, which may have led to insufficient power to demonstrate relationships precisely.	To examine health-related impairments in young children with ADHD and non-ADHD controls.	Children with ADHD had increased odds of moderate/large sleep problems (OR: 3.1; 95% CI 1.4, 6.8), compared with controls.
Williams and Sciberras (2016)	Longitudinal	ADHD: 112, ADHD symptoms: 648, No ADHD: 3349	75%/25%	68 children with ADHD on medication (MPH)	Birth-7 years	Sleep problems, emotional dysregulations and attentional regulation measured through mother reported specific questions, Strengths and Difficulties Questionnaire (SDQ). Limitations: Single-item measure of sleep problems does not allow for specificity in identifying particular sleep behaviors that contribute most to daytime functioning and development of self-regulation skills over time, relying on mothers' report alone leads to data being confounded with parental mental health and their own experience of ADHD symptoms.	To examine mean level differences and longitudinal and reciprocal relations among behavioral sleep problems, emotional dysregulation, and attentional regulation across early childhood for children with and without ADHD at 8 to 9 years.	Sleep problems in children with and without ADHD are associated with emotional dysregulation and poorer attentional functioning (between sleep problems at 0–1 year and emotional dysregulation at 0–1 year, $r = .37$, between sleep problems at 2–3 year and emotional dysregulation at 2–3 year $r = .18$, between sleep problems at 4–5 year and emotional dysregulation at 4–5 year $r = .21$, between sleep problems at 6–7 year and emotional dysregulation at 6–7 year, $r = .23$ ($p < .05$) and between sleep problems at 4–5 year and attention regulation at 4–5 year, $r = .14$ ($p < .05$).

(continued)

Table 1. (continued)

Authors/ publication year	Study design	Participant characteristics				Primary measures, limitations and biases	Objectives	Summary of main results
		Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age			Interventions/comparisons/ outcomes
Hansen et al. (2013)	Cross sectional longitudinal	Clinical grp.: 105 (out of which ADHD: 39, ADHD+Anx.: 25, Anx.: 41), control: 31	65.8%/34.3%	Not currently medicated.	7–13 years	semi-structured interview, Kaufman Schedule for Affective Disorders and Schizophrenia, CSHQ. Limitations: Small sample size, leading to lack of statistical power, lack of information regarding pubertal (pubertal onset related to sleep problems), use of only parent's report of sleep problems and not the child's report or objective sleep measures.	To examine the persistence and predictors of sleep problems over 18 months in children with anxiety disorders and/or ADHD and non referred controls.	Sleep problems may run a chronic course among children with ADHD (Restless sleep persisted in 32 children (persistence rate 72.7%), enuresis in 4 (persistence rate 66.7%), sleep talking in 22 children (persistence rate 68.8%), sleepwalking in 4 (persistence rate 57.1%), bruxism in 9 (persistence rate 60.0%), nightmares in 11 (persistence rate 50.0%) and night terrors in 2 (persistence rate 40.0%).
O'Callaghan et al. (2010)	Longitudinal	4204 parent responses	51.7%/48.3%	Not currently medicated.	5–14 years	Subjective questions for sleep problems, CBCL. Limitations: all reports of sleep and attention were through maternal reports and as such may be subject to bias, loss to follow-up may have led to biased estimates, without supportive clinical measures.	To examine whether sleep problems in infancy and early childhood are independently related to attention difficulty at 5 and 14 years, and to the continuity of attention difficulties from 5 to 14 years.	Sleep problems experienced "often" in early childhood are an indicator of subsequent attention problems that may persist into adolescence (odds ratio at 95% confidence interval, 1.84 (1.25, 2.70).
Gomes et al. (2014)	Case control	Hyperkinetic disorder: 30, Control: 30	83.3%/17.6%	13 children with ADHD on medication (MPH)	5–13 years	Portuguese Wechsler Intelligence Scale for Children, 3rd edition (WISC-III) or the Portuguese Wechsler Preschool and Primary Scale of Intelligence-Revised (WPPSI-R), Child Sleep-Waking questionnaire (CSWQ). Limitations: Sleep questionnaire did not address restless leg syndrome(RLS), limited clinical sample size leading to low statistical power, HKD sample followed ICD-10 criteria rather than DSM, children were not diagnosed through structured interviews, but instead according to a routine clinical evaluation by a child and adolescent psychiatrist.	To compare the parent-reported sleep of children with ICD-10 hyperkinetic disorder (HKD) versus community children.	ADHD)/HKD children may thus have more sleep-related problems than typically developing children (bedtime school nights median (mean)=21.30 (21.47) vs. 21.30 (21.12), $p<.01$; bedtime school nights median (mean)=21.30 (22.34) vs. 22.00 (21.56), $p<.05$; sleep length school nights median (mean)=09.30 (09.07) vs. 10.00 (09.46), $p<.01$; sleep length weekend nights median (mean)=09.30 (09:38) vs. 10:00 (10:15), $p<.05$; willingness to go to bed median (mean)=2.00 (2.33) vs. 4.00 (3.47), $p<.001$; bedtime refusal median (mean)=2.00 (2.30) vs. 1.00 (1.27), $p<.001$ measured through parent reports.
Becker et al. (2016)	Cross sectional	ADHD-I: 147	59%/41%	6 children with ADHD on medication (MPH-washout period 1 week before study commenced)	6–11 years	Child Symptom Inventory-4, K-SADS, Parent Inventory of Children's sleep habits. Limitations: All measures were completed by parents, the sleep measure used not been psychometrically validated, and objective measures of sleep (e.g., actigraphy, polysomnography) were not obtained, laboratory testing of endocrinological or metabolic factors that could cause/contribute to SCT/sleep disturbance were not conducted.	describe the sleep habits of children diagnosed with ADHD Predominantly Inattentive Type (ADHD-I), and (2) examine whether comorbid internalizing, oppositional, and/or sluggish cognitive tempo (SCT) symptoms are associated with poorer sleep functioning in children with ADHD-I.	Comorbid anxiety, in addition to sleepy/ tired symptoms, were most consistently associated with poorer sleep functioning in children with ADHD-I (beta coefficient of .35 for Anxiety for poor sleepers @ $p<.001$ and beta coefficient of .20 for sluggish cognitive tempo for poor sleepers @ $p<.05$, R squared –.20).

(continued)

Table 1. (continued)

Authors/ publication year	Study design	Participant characteristics: Sample size and diagnosis	Participant characteristics: Gender (male%/ female%)	Participant characteristics: Medication use	Participant characteristics: Age	Primary measures, limitations and biases	Objectives	Summary of main results: Interventions/comparisons/ outcomes
Hvolby et al. (2009)	Case control	ADHD: 45, control: 212, other psychiatric diagnoses: 64	82.2%/17.8%	Stimulant medication naïve.	5–11 years	Children Sleep Behavior Scale, K-SADS-PL. Limitations: parents not asked whether they view child's sleep difficulty as a sleep disorder, information regarding symptoms of breathing problems (SBD) or restless leg syndrome, possible psychiatric symptoms or ADHD-like behavior in the healthy control group were not assessed.	to describe sleep patterns and problems of 5–11-year-old children with ADHD described by parental reports and sleep questionnaires.	The ADHD group report problems with bedtime resistance, problems with sleep onset latency (in minutes) mean and SD, 44.3 (21.4) vs. 24.8 (11.1) $p < .001$, unsettled sleep and nightmares more often than the control groups.
Hansen et al. (2011)	Case control	Anxiety disorder: 41, ADHD: 39 comorbid condition:25), controls: 36	76.9%/23.1	Not currently medicated.	7–13 years	K-SADS, CSHQ. Limitations: Small sample size, only mother report, not self-report or sleep diary, was used to assess sleep problems, and the questionnaire assessed only the most recent typical week, diagnoses based on parent interview, and this could lead to under-reporting of anxiety symptoms in children with ADHD as well as ADHD symptoms in children with anxiety disorders.	To compare sleep problems in the referred children diagnosed with an anxiety disorder, ADHD, comorbid anxiety disorder and ADHD, and non-referred controls.	Night waking was associated with comorbid anxiety disorder and ADHD more than control (mean,CI)- 4 (3–8) vs. 3 (3–5), $p < .001$, effect size .67); bedtime resistance was associated with anxiety disorder, more than control (mean,CI)- 8 (6–17) vs. 6 (6–14), $p < .001$, effect size .43), while daytime sleepiness affected all clinical groups (mean,CI)- 12 (6–20) vs. 9 (6–17), $p < .01$, effect size .35, for ADHD>CTRL).
Rodopman-Arman et al. (2011)	Case control	ADHD: 40, control: 40	80%/20%	Not currently medicated.	7–13 years	CSHQ. Limitations: Subjective measures of parental reports were used rather than objectively collected sleep measures, lower incidence level for ODD comorbidity in sample, patients with psychiatric comorbidity excluded.	To investigate the sleep habits, associated parasomnias and behavioral symptoms in primary school children with ADHD.	Children with ADHD had more problematic sleep habits and night-time associated behaviors than controls. For example, sleep latencies of children with ADHD during the weekdays (21.6 ± 22 vs. 15.8 ± 12.8 (in minutes ± SD), p: .03) and on weekends (21.6 ± 19.1 vs. 14.9 ± 11.7 (in minutes ± SD), p: .001) were longer than in the normal controls. Similarly, rates of nightmares (p: .001), nocturnal enuresis (p: .001), habitual snoring (p: .007), and restless sleep (p: .02), daytime sleepiness ($p < .001$) were higher in children with ADHD.
Moreau et al. (2013)	Cross sectional	ADHD:43	58.1%/41.9%	31 children with ADHD on medication (extended and immediate release psychostimulants, Atomoxetine and psychostimulants + Atomoxetine.	6–13 years	Conner's Continuous Performance Test (CPT), Actigraph, Sleep diary, CSHQ, Conner's parent rating scale, K-SADS, BRIEF (Behavior Rating Inventory of Executive Functions. Limitations: Small sample size, heterogenous in terms of medication use, half of sample had inattentive subtype only, due to cross-sectional nature, causality between sleep disturbances and daytime functioning measures cannot be inferred.	To investigate potential relationships between two measures of sleep impairments (i.e., sleep duration and sleep efficiency [SE]) and attention and executive functioning in children with ADHD.	Shorter sleep duration was associated with a range of executive functioning problems as reported by the parents ((beta coefficient of .60 for total sleep time for behavioral regulation @ $p < .001$, R squared = .38 and Beta coefficient of .63 for total sleep time for global executive composite score @ $p < .001$, R squared = .29).

(continued)

Table 1. (continued)

Authors/ publication year	Study design	Participant characteristics: Sample size and diagnosis	Participant characteristics: Gender (male%/ female%)	Participant characteristics: Medication use	Participant characteristics: Age	Primary measures, limitations and biases	Objectives	Summary of main results: Interventions/comparisons/ outcomes
Akinci et al. (2015)	Case control	ADHD: 28, control: 15	71.4%/28.6%	Medication naïve.	8–12 years	PSQI (Pittsburgh Sleep Quality Index), Epworth Sleepiness Scale (ESS), Polysomnography (PSG). Limitations: Small sample size, PSG done for 1 night only.	To evaluate basic sleep architecture and non–rapid eye movement (NREM) sleep alterations in ADHD children.	Children with ADHD had worse sleep quality (Sleep efficiency, mean, CI, 98.4 (98–98.5) vs. 91.66 (87.16–95.22) @ $p < .001$) and more daytime sleepiness (ESS Score: mean, CI, 1 (0–2) vs. 3 (1.25–5) @ $p < .05$). Polysomnography data showed that the sleep macrostructure was not significantly different however, sleep microstructure was altered in ADHD children by means of reduced total cyclic alternating pattern rate (mean, CI, 61.1 (37.8–66.35) vs. 48.95 (19.25–59.65) $p < .031$) and duration of cyclic alternating pattern sequences (mean, CI, 6 (4.05–7.7) vs. 3.95 (3.2–6.75) $p < .044$).
Andersson and Sonnesen (2018)	Case control	ADHD: 15, control: 36	66.6%/ 33.4%	ADHD children on medication (no details given).	9–12 years	Intra-oral scans, Epworth Sleepiness Scale, Berlin Questionnaire. Limitations: small sample size and some aspects of dental and palatal dimensions could not be measured.	To compare sleepiness, occlusion, dental arch and palatal dimensions between children with ADHD and healthy children.	The ADHD children snored significantly more ($p < .05$) and slept restlessly significantly more often compared to the controls ($p < .001$) and had a tendency to sleep fewer hours during the night ($<.1$) and felt inadequately rested in the morning compared to the controls ($p < .1$).
Scott et al. (2013)	Longitudinal	ADHD: 173, total: 8195	84.4%/ 26.6%	No details given for medication use among ADHD diagnosed children.	6 months-11 years	Subjective created questionnaires for parents, consisting specific questions for sleep and other studied variables. Limitations: missing data and loss to follow-up more likely in socioeconomically deprived groups, psychosocial problems in family not addressed, might be associated with poor sleep and ADHD in children, information on children prescribed stimulant or other medication for their ADHD not reported accurately, subjective (parental reports) measures of sleep patterns and sleep behaviors.	To investigate sleep patterns and trajectories from 6 months after birth to 11 years old, and their relation to ADHD diagnoses.	In children with attention deficit hyperactivity disorder, shorter sleep duration appears early and predate the usual age of clinical diagnosis (at 6 months, mean:10 hour 35 minutes, (SD 1 hour 24 minutes) vs. mean; 10 hour 48 minutes (SD 1 hour 19 minutes) $p = .04$) and (at 6 years 9 months, mean:10 hour 50 minutes, (SD 0 hour 51 minutes) vs. mean; 11 hour 8 minutes (SD 0 hour 40 minutes) $p < .001$) as compared to controls.

(continued)

Table 1. (continued)

Authors/ publication year	Study design	Participant characteristics: Sample size and diagnosis	Participant characteristics: Gender (male%/ female%)	Participant characteristics: Medication use	Participant characteristics: Age	Primary measures, limitations and biases	Objectives	Summary of main results: Interventions/comparisons/ outcomes
Reynaud et al. (2018)	Longitudinal	1342 children (assessed at 2,3,5,6 years)	53%/47%	Not currently medicated.	2–6 years	Strength and difficulties questionnaire. Limitations: measurements for night-waking and inattention/ hyperactivity were both assessed through subjective questionnaire measures, lack of statistical power in the analysis of adjusted associations between covariates and the joint trajectories.	To study the longitudinal associations between inattention/hyperactivity symptoms and night-waking in preschool-years.	Both night-waking and inattention/ hyperactivity trajectories showed persistence of difficulties in preschool years (frequent night-waking trajectories ($N=1076$, 80%) and ("rare night-waking" one $N=269$, 20%), (high $N=174$, 13%), medium ($N=538$, 40%) and low ($N=630$, 47%) for inattention/hyperactivity z-score trajectories). Additionally, probability of having a high inattention/ hyperactivity trajectory when belonging to the common night-waking trajectory was of .20, vs. .13 ($p=.01$) when belonging to the rare one.
Touchette et al. (2009)	Longitudinal	2057 children	68.3%/34.3%	N/A	1.5–5 years	Subjective questionnaires assessing, sleep duration, hyperactivity scores, parental behaviors around sleep periods and potential risk factors, Infant characteristics questionnaire, Diagnostic Interview schedule for parents. Limitations: correlational design does not allow causal inferences, mother's perceptions of her child could have contributed to bias in the findings, most participants were non-immigrant French-speaking whites, the findings should be replicated in other populations.	To investigate the developmental trajectories of night-time sleep duration and hyperactivity over the preschool years and to identify the risk factors associated with short nighttime sleep duration and high hyperactivity scores.	Children with low hyperactivity scores are most likely to present an 11-hour persistent night-time sleep-duration trajectory (probability = .36) and children with high hyperactivity scores are most likely to present a short-persistent night-time sleep-duration trajectory (probability = .35). A χ^2 test revealed that night-time sleep-duration and hyperactivity trajectories were significantly associated ($\chi^2=75.1$; $p<.001$).
Tso et al. (2019)	Longitudinal	514 children	48.5%/51.5%	20 children received stimulant medication (MPH)	5–9 years	Chinese Childhood Sleep Quality Index (adapted from Children's Sleep Habits Questionnaire and the Hong Kong Adolescent Sleep Questionnaire), parent-rated Chinese Strengths and Weaknesses of ADHD-Symptoms and Normal-Behaviors (SWAN) questionnaire. Limitations: children's sleep patterns/quality of sleep based on parental reports rather than objective assessments, presence of ADHD symptoms measured via parent-rated questionnaire (not clinician assessments), some missing data due to attrition, impaired parent–child relationship may confound the association found, sample size is relatively limited in detecting effect of sex, despite observed effect size difference.	To study prospectively specific sleep patterns and risk of ADHD after adjusting for potential confounders such as obstructive sleep apnea (OSA) and methylphenidate use.	Sleep deprivation in early childhood is associated with higher risk of ADHD in middle childhood (the risk of probable ADHD was 15.5 per 100 for children with <8 hour of sleep in K3, whereas it was 1.1 per 100 for children with 11–12 hour of sleep in K3. The adjusted risk ratio was 14.19 ($p=.02$) (adjusted for obstructive sleep apnea and methylphenidate use).

(continued)

Table 1. (continued)

Authors/ publication year	Study design	Participant characteristics				Primary measures, limitations and biases	Objectives	Summary of main results
		Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age			Interventions/comparisons/ outcomes
Gregory et al. (2017)	Longitudinal	2042 Childhood ADHD (ADD screened at 5,7,8,10,12 & 18 years)	49%/51%	Participants taking ADHD medication excluded from analysis.	5–18 years	PSQI, ADHD diagnostic tools. Limitations: Self-reports of ADHD and sleep quality may have artificially inflated the associations, collected information on sleep quality in young adulthood, but not prior during childhood.	To examine the longitudinal association between ADHD and sleep quality and to explore the genetic and environmental underpinnings of this association.	Children with ADHD had poorer sleep quality in young adulthood ($\beta = .19$ [95% CIs] = .14, .25, $p < .001$), but only if their ADHD persisted. Adults with ADHD had more sleep problems than those without ADHD, over and above psychiatric comorbidity and maternal insomnia (hyperactive/impulsive $\beta = .13$ [95% CIs] = .07, .19, $p < .001$; inattentive $\beta = .20$ [95% CIs] = .14, .25, $p < .001$).
Grünwald and Schlarb (2017)	Cross sectional	ADHD: 72	79.2%/20.8%	Medication naïve.	6–13 years	CSHQ- German version, Quality of life questionnaire, parent rated German language questionnaires for diagnosis of ADHD (FBB-HKS). Limitations: Small sample size, gender disparity with more male participants, no objective measurements included.	To examine the links between sleep disorders and subtypes of attention deficit-hyperactivity disorder (ADHD-inattention, ADHD-combined, ADHD-hyperactive/ impulsive) in childhood.	Children with primarily hyperactive-impulsive ADHD showed the highest CSHQ-DE scores (parasomnias: ADHD-I, 1.09 (.11); ADHD-H, 1.38 (.31); ADHD-C, 1.26 (.28) ($\chi^2[2] = 6.343$; $p = .042$) sleep-disordered breathing: ADHD-I, 1.00 (.00), ADHD-H, 1.10 (.16), ADHD-C, 1.17 (.29) ($\chi^2[2] = 6.107$; $p = .047$) and high impact for insomnia in this subgroup ADHD-I ($N = 0$; .0%), ADHD-HI ($N = 6$; 50.00%), and ADHD-C ($N = 6$; 31.58%) and a high comorbid load for the mutual occurrence of insomnia and nightmares.
Bessey, Coulombe, et al. (2013)	Case control	ADHD: 84, control: 179	Not mentioned	Not mentioned	5–12 years	Sleep Attitudes and Belief Scale (SABS). Limitations: Samples did not significantly differ on most demographic variables, allowing valid between-group comparisons, age of target children were not collected, ADHD/ASD current status of the child could not be determined.	To develop a measure to assess parental beliefs about their child's sleep as a contributing factor for typically developing, ADHD and ASD children.	As compared to the TD children's parents, both ADHD (sleep modifiability subscale: t (352) = –5.60, $p < .001$; and responsiveness to treatment subscale: t (352) = –2.99, $p = .003$) and ASD (sleep modifiability subscale: t (352) = –4.27, $p < .001$; and responsiveness to treatment subscale: t (352) = –3.04, $p = .005$),children's parent's reported that their child's sleep problems are less modifiable and responsive to change.
Matsuoka et al. (2014)	Case control	Developmental disorders: 43 (out of which ADHD:9), control: 372	90.7%/9.3% (developmental disorders group)	In developmental disorder group, 9 children were on medication (MPH, antipsychotic drugs, antiallergic drugs), in control group 30 on medication (antipsychotic drugs, antiallergic drugs, anti-epileptic).	6–12 years	CSHQ, PSQI. Limitations: Reliability/ validity of used Japanese CSHQ not determined by time of administration and scoring, medicated children with developmental disorder had higher total CSHQ and sleep onset delay scores than those who were not on medication, suggesting medications may have adverse effects on sleep, only subjective reports of questionnaires used to assess sleep and small female sample.	To analyse the sleep problems of children with developmental disorders, such as pervasive developmental disorder and attention deficit hyperactivity disorder.	The total CSHQ score ($r = .429$, $p < .001$), bedtime resistance ($r = .376$, $p < .05$), sleep onset delay ($r = .372$, $p < .05$), and daytime sleepiness worsened ($r = .463$, $p < .05$) with increasing age in children with developmental disorders; in contrast, these parameters were unchanged or became better with age in the control group. In children with developmental disorders, there was a significant association between a higher total CSHQ score and lower academic performance ($r = .37$, $p < .05$).

(continued)

Table 1. (continued)

Authors/ publication year	Study design	Participant characteristics				Primary measures, limitations and biases	Objectives	Summary of main results
		Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age			Interventions/comparisons/ outcomes
Noble et al. (2011)	Cross sectional	ADHD: 67	70%/30%	10 children with ADHD were on medication (stimulants or Atomoxetine).	5–12 years	Conners Questionnaire, BACS, parenting stress index, impairment rating, CPRS, Child Routines Inventory (CRI)–Parent Report Form, CSHQ. Limitations: Objective measures not used, possibly leading to rater's bias, all aspects of parenting factors not captured, no information about direction of influence between predictor variables and sleep difficulties in children.	To examine the extent to which parental influence predicted sleep problems among ADHD children.	Majority of parents/caregivers (73%) reported significant child sleep difficulties. Parental implementation of daily routines added to the explained variance in bedtime resistance after considering child and family characteristics (explaining 25% of the variance in CSHQ scores, F (4; 62) = 5.21, $p < .01$).
Sciberras et al. (2017)	Cross sectional	ADHD: 361	75%/25%	268 children with ADHD on medication (stimulants), Melatonin (124), Clonidine (31).	6–12 years	CSHQ, Anxiety Disorders Interview schedule, Subjective questions for parents assessing sleep hygiene and parenting consistency and warmth, parental mental health assed through Kessler-6 (K-6). Limitations: Only subjective measures used, no valid measures of sleep hygiene, absence of non ADHD control group.	To examine the association between sleep problems and parenting and sleep hygiene in children with ADHD.	Sleep hygiene and parenting are important modifiable factors independently associated with sleep problems in children with ADHD (greater parenting consistency was associated with decreased bedtime resistance (R squared = .14) and decreased sleep anxiety (R squared = .08), and with increased delayed sleep onset (R squared = .08) & (poorer sleep hygiene was associated with increased bedtime resistance (R squared = .14), increased daytime sleepiness (R squared = .16), increased sleep duration problems (R squared = .12), and increased delayed sleep onset (R = .08).
Kwon et al. (2014)	Cross sectional	ADHD:56 (with RLS 24, without RLS 32)	91%/9%	All ADHD participants were on medication.	Mean age 10.7 years	Sleep questionnaire and Restless leg syndrome questions. Limitations: Influence of drug therapy not excluded, objective tests such as PSG or iron workup not performed, small sample and absence of a control to compare, comparable number of female participants were not included in the sample.	To identify the prevalence of RLS and sleep problems in children with ADHD in Korea.	12.5% had a family history of RLS, 42.9% showed symptoms of RLS, (out of which 3.6% were diagnosed with definite and probable RLS based on sleep questionnaire), 45% of patients had behavioral insomnia, 45% body movements, 30% teeth grinding, 21% kicking legs, unrefreshed sleep 77%, looking tired 50%.

2010; Eyuboglu & Eyuboglu, 2018). Additionally, subjective reports by parents revealed that sleep problems emerging early for children (aged 6–8 years) with ADHD ($n = 177$) are uniquely associated with co-occurring internalizing and externalizing behaviors (Sciberras et al., 2016).

Williams and Sciberras (2016) report in a longitudinal study that children with mother-reported ADHD symptomatology had significantly more behavioral sleep concerns, along with poorer emotional and attentional regulation, and these features were evident from 2 to 3 years and remained poorer up to the age of 6 to 7 years (ADHD $n = 112$, ADHD symptomatic $n = 648$). In another longitudinal study of 5 to 14 year-old children, 4204 responses revealed parent-reported sleep problems "often" were independently associated with early remitter and persistent attention problems, and "sometimes" associated with early remitter and adolescent onset attention problems (O'Callaghan et al., 2010). Persistence of parent-reported sleep problems with usual treatment after diagnosis was found not to differ for 39 children with ADHD (7–13 years) when measured at 2 time points (within 18 months, through the CSHQ) with parent ratings above the clinical cut off score of 41 at both time points (Hansen et al., 2013), indicating that sleep problems may run a chronic course among children with ADHD.

Parent/caregiver-reported presence of sleep-related psychological problems have been found among children with ADHD ($n = 30$, aged 5–13 years), such as more incidence of anxiety, nightmares and needing the bedroom lights to be on (Gomes et al., 2014). Sluggish cognitive tempo was found to be associated with shorter sleep duration, poorer sleep, being harder to wake and becoming alert after waking in the morning among 147 ADHD-inattentive children aged 6 to 11 years (Becker, Pfiffner, et al., 2016). Hvolby et al. (2009) found that 13.3% of all children with ADHD (45) have nightmares, compared with only 1.4% of healthy children (212). It is also important to note here that parental ratings for 7 to 13-year-old children with ADHD comorbid with anxiety disorder ($n = 25$) revealed not only higher scores for CSHQ, but also higher occurrence of sleep anxiety and night waking (Hansen et al., 2011). Rodopman-Arman et al. (2011) found that among their ADHD sample of children ($n = 40$), about 22% (vs. 2.9% of the control group) needed their parents to accompany them while going to sleep, and transitional objects were needed by 8.1% of children with ADHD in contrast to 2.9% of controls ($n = 40$).

Considering parent-reported sleep latency and total obtained sleep, significant differences have been observed in children with ADHD. In a group of 147 ADHD-predominantly inattentive (ADHD-I) children (aged 6–11 years), 14 percent were reported to achieve less sleep than recommended age-appropriate duration (7.5–11 hours), and 31% showed sleep onset latency greater than 20 minutes (Becker, Froehlich, et al., 2016). Similar findings were also reported by Hvolby et al. (2009) when 31% of children with ADHD ($n = 45$) were reported to be unwilling to go to bed, 22% had difficulty falling asleep and a greater proportion of children with ADHD had long sleep latency time compared to healthy controls ($n = 212$) and a non-ADHD psychiatric group ($n = 64$). Moreau et al. (2014) found that 41 children with ADHD (both medicated and un-medicated) had a higher score than the control group ($n = 41$) for the scale of Insomnia Severity Index for children (ISI-C).

Poorer ratings for sleep quality, duration and efficiency have also been reported for children with ADHD. Parents reported poorer total sleep quality scores on the Pittsburgh Sleep Quality Index (PSQI) for 28 children with ADHD (8–12 years) along with higher sleep latency and lower reported sleep efficiency when compared to the typically functioning group (Akinci et al., 2015). In this same study, increased scores for daytime sleepiness among the ADHD group were also reported. Children with ADHD ($n = 15$) have been reported to achieve shorter sleep duration and to be more restless in bed compared to controls ($n = 36$) (Andersson & Sonnesen, 2018). A longitudinal study of an ADHD cohort ($n = 173$) found a consistent decrease in sleep duration by one standard deviation to be a significant predictor of ADHD at 3 to 5 years; additionally, night waking was significantly present from the age of 5 years through to 9 years 7 months (Scott et al., 2013). Reynaud et al. (2018) found that both parent-reported night waking and inattention/hyperactivity trajectories were strongly correlated between 2 and 5 to 6 years ($n = 1{,}324$). Even for a younger age group, longitudinal study (ages 1.5 to 5 years, using self-administered questionnaires for the mothers of 2057 children), night time sleep duration and hyperactivity trajectories were highly inter-correlated (Touchette et al., 2009). The same study also found that children with shorter sleep duration and high hyperactivity scores were boys from low income families and with mothers having low educational attainment. The link between shorter sleep duration and ADHD symptomatology was also reported when 411 parent–child dyads were assessed at two time points (Kindergarten and primary 3 in China, 5–9 years), revealing a higher risk of probable ADHD in primary 3 (as measured through parent rated questionnaire and DSM-V) for children who slept less than 8 hours during kindergarten, as opposed to those children who slept between 11 and 12 hours during kindergarten (Tso et al., 2019). Here the authors also found a higher prevalence of probable ADHD among children who had 0 to 3 days of good quality sleep (during primary 3) as compared to the children who had 6 to 7 days of good quality sleep. A longitudinal analysis from childhood to early adulthood ($n = 2{,}042$) in a twin cohort (ADHD diagnoses at 5,7,10,12 and 18 years-old) revealed that children with ADHD had poorer sleep quality in young adulthood (self-rated by patient), but only if their ADHD persisted (Gregory et al., 2017).

Particular trends of parental reported sleep problems have been identified for individual ADHD subtypes. As compared to inattentive subtype, ADHD hyperactivity subtype children received elevated ratings on the CSHQ (Eyuboglu & Eyuboglu, 2017; Grünwald and Schlarb, 2017). Grünwald and Schlarb (2017) also noted higher comorbid occurrence of insomnia and nightmares in this sub group (6–13 years, $n=72$). Subjective reports by parents revealed higher prevalence of sleep problems in children with ADHD combined presentation (30%) as compared to ADHD inattentive presentation (17%), however this finding was not statistically significant (Sciberras et al., 2016). However, in a previous study by Hansen et al. (2011), ADHD combined and hyperactive subtype ratings did not differ from ADHD inattentive on the CSHQ scale (7–13 years, $n=39$).

Bessey, Richards, et al. (2013) studied parental attitudes and beliefs with regard to their ADHD child's sleep as compared to those held by parents of typically developing children. In this study, 84 respondents on the Sleep Attitudes and Beliefs Scale (SABS) were parents of children with ADHD, 92 of ASD (Autism Spectrum Disorder) children and 179 were parents of typically developing children (all children 5–12 years). Results indicated that compared to the TD children's parents, both ADHD and ASD children's parents reported that their child's sleep problems are less modifiable and responsive to change. A number of studies have focused on parent's own level of functioning and daily behaviors that might be associated with their children's sleep problems. Matsuoka et al. (2014) found a higher significant positive correlation between parent reported total CSHQ scores for their ADHD/ASD children ($n=43$, 6–12 years) and their own sleep quality's self-report using the Pittsburgh Sleep Quality Index (PSQI) when compared to parents of typically functioning children. In a cross sectional study among 67 children with ADHD (5–12 years) Noble et al. (2011) found that parental-reported lack of consistent daily routine was a significant predictor for their child's bedtime resistance and along with that parenting stress was negatively correlated with daily implemented living routines and also predicted sleep anxiety for the child with ADHD. Additionally, parent implemented daily living routine was correlated with the family's income and lower income parents tend to implement less consistent daily routines. Consistent with these findings, Sciberras et al. (2017) found that for 361 children with ADHD (5–13 years) greater parenting consistency and better sleep hygiene were associated with decreased bedtime resistance, while better sleep hygiene was associated with lower levels of daytime sleepiness, less delayed sleep onset (associated with greater parental warmth), and fewer sleep duration difficulties.

Objectively measured macro-structural properties of sleep among children with ADHD. Several studies have utilized actigraphic measures for the estimation of total sleep time and sleep latency among children with ADHD (studies detailed in Table 2). Case control investigations have revealed shorter sleep duration and longer sleep latency among children with ADHD compared to the typically developing control group (Lee et al., 2014; Miano et al., 2019; Moreau et al., 2013). Moreau et al. (2013) also found that children with ADHD ($n=43$, 6–13 years) with comorbid psychiatric conditions had significantly longer sleep onset latency when compared to the ADHD-only group or the control group. When total sleep time is considered it was also found that children with ADHD with comorbid conduct disorder ($n=16$) slept significantly less than children in the control group ($n=30$) and those with only conduct disorder (CD= 4) as measured through actigraphy (difference of −50 minutes; Aronen et al., 2014). Contrary to the above findings, Wiebe et al. (2013), Bergwerff et al. (2016) and Waldon et al. (2018) found no significant difference in actigraphy-measured total sleep time and sleep latency for children with ADHD as compared to typically developing control groups. The study by Waldon et al. (2018) also found that sleep duration significantly predicted performance on an objective attention test in both children (6–12 years) with ADHD ($n=25$) and TD ($n=25$) children, but did not significantly predict parent-reported attention supporting that poor sleep would predict poor attention regardless of whether or not the child had ADHD (Waldon et al., 2018). For night-to-night variability in actigraphic sleep parameters among 7 to 12-year-old ADHD ($n=50$) and typically developing children ($n=50$), significant difference in sleep duration was only found among the different nights, but the difference was not significant between the ADHD and the control group (Poirier & Corkum, 2018). However, if the presence of comorbid CD/ODD is considered, Bergwerff et al. (2016) found that for time in bed, significant interaction effects were found between the aggregated ADHD measure and parent and teacher rated CD and Oppositional defiant disorder (ODD) ($n=63$, 6–13 years); similarly, for total sleep time significant interactions between the aggregated ADHD measure and parent and teacher rated internalizing and CD/ODD behavior.

For sleep efficiency quantitated through actigraphy recordings, children with ADHD ($n=43$, 6–13 years) were found to have significantly lower sleep efficiency compared to healthy control group children (Moreau et al., 2013). Children (7–12 years) with ADHD and comorbid conduct disorder ($n=16$) had significantly lower sleep efficiency than both children in the control group ($n=30$) and those with only conduct disorder ($n=14$) (Aronen et al., 2014). However, Lee et al. (2014) found no significant difference in sleep efficiency between the ADHD group ($n=37$) and typically developing peers ($n=32$) in the same age group. When sleep actigraphy results at home and sleep lab were compared for 25 children with ADHD (6–12 years) with age

Table 2. Summary of Nine Studies Included Focusing on Macro-Structural Sleep Properties Among ADHD Children.

Authors/ publication year	Study design	Participant characteristics				Primary measures, limitations and biases	Objective	Summary of main results
		Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age			Interventions/comparisons/outcomes
Lee et al. (2014)	Case control	ADHD: 37, control: 32	100%/0%	Medication naïve.	7–12 years	Matching Familiar Figures Test (MFFT) and 72-hour actigraphy. Limitations: Diagnosis was based on interview with no ADHD subtyping, small sample sizes, only male patients.	To examine neurocognitive functions and nocturnal sleep parameters in patients with ADHD, using a cognitive function test and actigraphy.	Patients with ADHD had more sleep problems, including significantly increased sleep latency (mean (SD) 16.2 (19.7) vs. 6.6 (6.2) $p = .01$), WASO (mean (SD) 57.4 (23.2) vs. 30.7 (13.7) $p < .001$), and fragmentation index (mean (SD) 16.7 (4.5) vs. 10.3 (4.4) $p < .001$), and poorer cognitive function (WASO measured by actigraphy positively correlated with the error rate of response measured by the MFFT in the ADHD patient group (rho = .52, $p = .012$)), compared with controls.
Miano et al. (2019)	Case control	ADHD: 30, control: 27	70%/30%	Medication naïve.	7.5 years-13.5 years	K-SADS-PL, NEPSY-II (Neuropsychological Test-Second edition), WISC-V, nocturnal video PSG, actigraphy, Multiple sleep latency test. Limitations: Controls did not undergo actigraphic and MSLT recordings, all children underwent only one PSG recording (first night effect not controlled) andactigraphic recording was performed 1 week before PSG and MSLT, or the week after, depending on the needs of the child, without checking for naps before PSG recording.	To describe the SWA behavior in the same group of children with ADHD.	The focus of SWA (Slow Wave Activity) was observed over the centro–parietal–occipital regions in participants with ADHD ((Permutation test, FDR-corrected, $p < .01$), which remained significant in the subgroups divided between subgroups according to the sleep diagnosis ($p < .01$).

(continued)

Table 2. (continued)

Authors/ publication year	Study design	Participant characteristics				Primary measures, limitations and biases	Objective	Summary of main results
		Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age			Interventions/comparisons/outcomes
*Moreau et al. (2013)	Cross sectional	ADHD: 43	58.1%/41.9%	31 children with ADHD on medication (extended and immediate release psychostimulants, Atomoxetine and psychostimulants + Atomoxetine.	6–13 years	Conner's Continuous Performance Test (CPT), Actigraph, Sleep diary, CSHQ, Conner's parent rating scale, K-SADS, BRIEF (Behavior Rating Inventory of Executive Functions). Limitations: Small sample size, heterogenous in terms of medication use, half of sample had inattentive subtype only, due to cross-sectional nature, causality between sleep disturbances and daytime functioning measures cannot be inferred.	To investigate potential relationships between two measures of sleep impairments (i.e., sleep duration and sleep efficiency [SE]) and attention and executive functioning in children with ADHD.	Shorter sleep duration was associated with a range of executive functioning problems as reported by the parents ((beta coefficient of .60 for total sleep time for behavioral regulation @ $p < .001$, R squared = .38 and Beta coefficient of .63 for total sleep time for global executive composite score @ $p < .001$, R squared = .29).
*Aronen et al. (2014)	Case control	CD & ODD: 30(16 comorbid ADHD), Control:30	90%/10%	11 children from the clinical group were on medication (Risperidone and MPH)	7–12 years	K-SADS, CSHQ, Actigraphy. Limitations: Small sample size, likely limiting the statistical power of the tests used, the actigraphic measurement time (3 nights) was only modest.	To compare the self- and parent-reported sleep problems and objectively measured sleep amount and efficiency in child patients with CD/ ODD and their age- and gender-matched controls.	Children with comorbid ADHD slept significantly less than did the patients with CD/ODD alone and the controls (total sleep minutes mean ± SD = 500.3 ± 50.2 (ADHD+CD/ODD) vs. 416.3 ± 59.8 (CD/ODD), $p < .001$ as measured through actigraphy).

(continued)

Table 2. (continued)

Authors/ publication year	Study design	Participant characteristics				Primary measures, limitations and biases	Objective	Summary of main results
		Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age			Interventions/comparisons/outcomes
Wiebe et al. (2013)	Case control	ADHD: 26, Control: 56	62%/ 38%	4 children with ADHD on medication (washout period of 48 hours before sleep assessment).	7–11 years	PSG, actigraphy, Multiple sleep latency test (MSLT), Epworth Sleepiness Scale (ESS). Limitations: Small sample size, pediatric norms not well established for the MSLT, restless legs syndrome was not measured.	To assess the association between habitual sleep patterns and one night of PSG measured sleep with daytime sleepiness in children with ADHD and typically developing children.	Actigraphy measures of sleep restlessness (time awake ($r = .48$) and activity ($r = .48$) during the night), as well as time in slow-wave sleep ($r = .46$), were positively related to mean sleep latency on the multiple sleep latency test in children with ADHD (@ $p < .05$).
Bergwerff et al. (2016)	Case control	ADHD: 63, Control: 61	52%/48%	39 children from the ADHD group were on medication (stimulants).	6–13 years	Actigraphy, CBCL & TRF, SES evaluation on 6-point Likert scale. Limitations: Used objective measure of sleep quality does not provide insight into parent–child interactions.	To gain more insight into sleep problems in ADHD using objective measures of sleep quality.	No objectively measured sleep parameters difference was found.
Waldon et al. (2018)	Case control	ADHD: 25, Control: 25	82%/18%	Medication naive.	6–12 years	Conners' Parent Rating Scale-Revised: Long Version, Attention Network Test-Interaction (ANT-I), Actigraphy. Limitations: Larger sample size would have allowed more in-depth analysis that may have permitted analyses examining differences in sex or ADHD presentations.	To examine the relationships between sleep and attention in both typically developing (TD) children and children with ADHD.	Children with ADHD had poorer alerting (689.96 (618.61) vs. 264.60 (341.97), $F(1, 48) = 9.05$, $p < .001$) and executive attention (1,121.44 (760.68) vs. 483.49 (820.88), $F(1, 48) = 8.12$ $p < .01$) on the ANT-I and poorer parent-reported attention (71.20 (9.38) vs. 47.64 (10.31), $F(1, 48) = 71.44$, $p < .00$), poor sleep predicted performance on alerting attention for children with ADHD and TD children, (R squared = .32, $F(1, 48) = 4.09$, Beta coefficient = −4.10, $p < .001$) whereas the interaction between poor sleep and ADHD diagnosis predicted executive attention scores (R squared = .33, $F(1, 48) = 4.25$, Beta coefficient = −5.14, $p < 001$).

(continued)

Table 2. (continued)

Authors/ publication year	Study design	Participant characteristics				Primary measures, limitations and biases	Objective	Summary of main results
		Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age			Interventions/comparisons/outcomes
Poirier and Corkum (2018)	Case control	ADHD: 50, Control: 50	82%/8%	Medication naive.	7–12 years	Actigraphy and sleep diaries for 4 nights. Limitations: Data collected over a number of years, used same method across studies in terms of ADHD diagnosis and actigraphy data collection, it is possible that different results are associated with different presentations of ADHD (sample included all three subtypes), small sample, larger number of male participants.	To examine the night-to-night variability of sleep between typically developing children and children with ADHD.	No night to night variability between the ADHD or TD children.
Bessey, Richards, et al. (2013)	Case control	ADHD: 25, Control: 25	88%/12%	Medication naive.	6–12 years	Conners' Rating Scale-R, Demographic Questionnaire,Mini-Motionlogger Actigraphs,Sleep Lab Adaptation Questionnaire (SLAQ). Limitations: Findings should be cautiously generalized from PSG to the home, 1 night sleep study, small sample, majority male participants in both groups.	To investigate sleep lab adaptation in children with ADHD and typically developing children, using actigraphy and parent–child-completed questionnaires.	ADHD children indeed did not sleep differently than TD children in lab, however sleep efficiency of TD children was more than ADHD children (for TD children, sleep efficiency was 79.90% (SD = 10.76%) at home and 86.11% (SD = 7.25%) in the sleep lab; the main effect (F (1, 48) = 5.79, P = .020) and the interaction were significant (F (1, 48) = 5.53, P = .023).

matched control group, both groups slept 50 minutes less in the sleep lab and sleep efficiency was improved for the typically developing group ($n=25$) in the sleep lab conditions, but not for children with ADHD (Bessey, Coulombe, et al., 2013).

Assessment of restlessness or movement during sleep measured through actigraphy has indicated increases in the sleep fragmentation index among children with ADHD in a number of studies. Wake after sleep onset (WASO) refers to periods of wakefulness occurring after defined sleep onset (Shrivastava et al., 2014) and has also been explored in a number of studies with children with ADHD. When compared to typically developing 7 to 12 years-old children ($n=32$), children with ADHD ($n=37$) were found to have more WASO and higher fragmentation index scores (Lee et al., 2014). For children with ADHD, error and response latency rate on an objective test of impulsivity was also positively correlated with the WASO and fragmentation index (Lee et al., 2014). Mean activity count per wakening epoch after sleep onset are also reported to be elevated in children with ADHD compared to typically developing children and higher still in children with ADHD and comorbid psychiatric condition. However, Bergwerff et al. (2016) found no significant difference in actigraphic measures of nocturnal motor activity for children with ADHD ($n=63$) as compared to typically developing control group ($n=61$).

Objectively measured micro-structural properties of sleep in children with ADHD. A number of studies have studied Rapid Eye Movement (REM) sleep characteristics measured by nocturnal polysomnography (PSG) in children with ADHD (studies detailed in Table 3). Akinci et al. (2015) found that 8 to 12-year-old children with ADHD ($n=28$) had a lower percentage of REM sleep compared to healthy controls ($n=15$). A home PSG also found significantly shorter duration of REM sleep, smaller percentage of total time spent in REM sleep and shorter sleep duration, in the ADHD group ($n=15$) compared to control group ($n=23$) children aged 7 to 11 years (difference of ~15min per night in REM sleep; Gruber et al., 2009). Díaz-Román and Buela-Casal (2019) report that similar age group children with ADHD ($n=20$) had shorter REM latency compared to typically developing children ($n=20$) and Grissom et al. (2009) found that 6 to 10-year-old children with ADHD ($n=13$) showed significantly lower frequency of eye movement during REM sleep compared to the control group ($n=16$).

For Non-REM (NREM) sleep properties in children with ADHD, Akinci et al. (2015) report lower cyclic alternating pattern (CAP; periodic EEG activity which recurs at intervals up to 2 minutes), proportion of sleep time spent in total NREM and in Stage 2 (N2) sleep along with reduced A1 index (high voltage slow wave EEG activity) in N2 as well as shorter sequence mean duration and lower CAP rate A1. The authors also found that children with ADHD ($n=28$, 8–12 years) had had lower SpO2 and NREM SpO2 (both being significantly correlated to the Sleep Quality subjective measure, PSQI). Grissom et al. (2009) found that children with ADHD ($n=13$, 6–10 years) had significantly lower percentage for stage 2 sleep when compared to controls and conversely that they had significantly higher percentage of stage 3 sleep when compared to controls. In line with the above findings, Silvestri et al. (2009) found significant differences in almost all of the studied sleep variables (REM%, N3% N2%, N1%, SE% (sleep efficiency), TST (total sleep time) MIN, REM Latency, SLEEP Latency) between 55 children with ADHD and 20 controls (6–12 years). Markovska-Simoska and Pop-Jordanova (2017) found children with ADHD have increased absolute power of slow waves (theta and delta) as compared to matched controls. When the power of slow sleep spindles during stage 2 NREM sleep was evaluated, Saito et al. (2019) found that the ratio of 12-Hz frontal spindle power was higher in 5 to 13-year-old ADHD ($n=21$) than in the typically developing children ($n=18$) (especially for children with ADHD with comorbid ASD), and also that the ratio of 12-Hz spindles was significantly correlated with reaction time variability on a CPT to measure attention. EEG power spectral analysis revealed significant differences concentrated in the period immediately after spindle epochs, in the left hemisphere of the brain, in almost all bands, with greater values in control ($n=7$) than in children with ADHD ($n=8$) among 6 to 10 year olds (De Dea et al., 2018).

Topographical distribution of Slow Wave Activity (SWA) recorded through high definition EEG nocturnal recordings in children with ADHD revealed a local increase of SWA in a cluster of six electrodes over central regions in 6 to 12-year-old children with ADHD ($n=9$) compared to control ($n=9$) children, indicating a less mature topographical SWA distribution in comparison to healthy children of the same age and sex (Ringli et al., 2013). In another study, full night high definition PSG assessment of children with ADHD revealed a decrease of SWA during the night, with a focus of SWA over the centro–parietal–occipital regions and greater delta power over the posterior cingulate in participants with ADHD ($n=30$, 7–13 years) (Miano et al., 2019). However, there is a level of inconsistency in the literature regarding sleep EEG findings in children with ADHD. Příhodová et al. (2012) found no significant differences in any of the CAP parameters between 7 and 12 year children with ADHD ($n=14$) and controls ($n=12$). On similar lines, in an earlier study, Choi et al. (2010) found no significant differences between ADHD and healthy groups in any of the polysomnographic sleep measures. Again, when studying the effect of stimulant medication (MPH) on sleep architecture in 27 children (6–12 years) with ADHD, Galland et al. (2010) found that sleep architecture was preserved and the arousal indices remained unchanged.

Table 3. Summary of Sixteen Included Studies Focusing on Micro-Structural Sleep Properties Among ADHD Children.

Authors/ publication year	Study design	Participant characteristics				Primary measures, limitations and biases	Objective	Summary of main results
		Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age			Interventions/comparisons/ outcomes
*Akinci et al. (2015)	Case control	ADHD: 28, Control: 15	71.4%/28.6%	Medication naïve.	8–12 years	PSQI (Pittsburgh Sleep Quality Index), Epworth Sleepiness Scale (ESS), Polysomnography (PSG). Limitations: Small sample size, PSG done for 1 night only.	To evaluate basic sleep architecture and non–rapid eye movement (NREM) sleep alterations in ADHD children.	Children with ADHD had worse sleep quality (Sleep efficiency, mean, CI, 98.4 (98–98.5) vs. 91.66 (87.16–95.22) @ $p < .001$) and more daytime sleepiness (ESS Score: mean, CI, 1 (0–2) vs. 3 (1.25–5) @ $p < .05$). Polysomnography data showed that the sleep macrostructure was not significantly different however, sleep microstructure was altered in ADHD children by means of reduced total cyclic alternating pattern rate (mean, CI, 61.1 (37.8–66.35) vs. 48.95 (19.25–59.65) $p < .031$) and duration of cyclic alternating pattern sequences (mean, CI, 6 (4.05–7.7) vs. 3.95 (3.2–6.75) $p < .044$).
Gruber et al. (2009)	Case control	ADHD: 15, Control: 23	66%/34%	Not currently medicated.	7–11 years	CBCL (Child Behavior Checklist), CSHQ & PSG. Limitations: nasal cannulas not used to detect obstructive hypopneas more accurately; and Multiple analyses leading to subsequent potential for type I errors.	To examine sleep architecture and reported sleep problems in children with ADHD and normal controls.	ADHD group had significantly shorter duration of REM sleep (ADHD-84.18 ± 32.73* vs. TD- 100.23 ± 24.99, $p < .05$).
Díaz-Román et al. (2018)	Case control	ADHD: 20, Control: 20	67%/33%	17 children with ADHD on medication (MPH, Atomoxetine, MPH+Atomoxetine).	7–11 years	Paediatric Daytime sleepiness Scale (PDSS), Paediatric Sleep Questionnaire (PSQ), sleep diary, PSG. Limitations: The impact of possible confounding variables namely, ADHD subtypes, medication, and SCT symptoms were not assessed.	To compare the subjective and objective sleep characteristics of children with ADHD and typically developing children.	No significant difference was found between the groups in almost any objective sleep variable, except for shorter REM latency in the ADHD group (ADHD-144.19 (53.53) vs. TD-195.70 (52.75), $p < .01$) and these children showed significantly higher levels of daytime sleepiness (ADHD-10.10 (5.98) vs. TD-5.25 (2.95), $p < .05$) and greater general sleep problems (ADHD-.30 (.17) vs. TD-.11 (.13), $p < .001$) than control children, as reported by their parents.
Grissom et al. (2009)	Case control	ADHD: 13, Control: 16	Not mentioned	Not mentioned	6–10 years	PSG. Limitations: Records from previously conducted PSGs were analyzed, thereby leaving behind possible gaps in full information regarding behavioral and environmental effects for the child during that time.	To examine eye movements during rapid eye movement (REM) sleep of children with ADHD and a typically developing control group.	The results indicate a pattern of higher amplitude ($U = 44.00$, $p < .01$), lower frequency REM sleep eye movement in children with ADHD (with an average rank of 7.62 vs. 21.00 for children with ADHD).

(continued)

Table 3. (continued)

Authors/ publication year	Study design	Participant characteristics				Primary measures, limitations and biases	Objective	Summary of main results
		Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age			Interventions/comparisons/ outcomes
Silvestri et al. (2009)	Case control	ADHD: 55, Control: 20	85%/15%	Not mentioned.	6–12 years	Conners and SNAP-IV Scales, a structured sleep interview and a nocturnal video-PSG. Limitations: PSG was done for only 1 night thereby not controlling the first night's effect in the results.	To examine specific sleep disturbances and sleep disorders in different clinical subsets of ADHD children.	There is a significant difference in almost all the studied sleep variables between ADHD children and controls (REM% N3%, N2%, SE%, MA INDEX, TST MIN, REM Latency @ $p < .05$) and different sleep disorders seem to address specific ADHD phenotypes (PLMS index- ADHD-I- 9.0 (22.0) vs. ADHD-C-14.5 (19.2) @ $p < .05$) and correlate with severity of symptoms (Restless leg syndrome severity: ADHD-I- 1.3 (5.1) vs. ADHD-C- 6.9 (10.4) @ $p < .05$).
De Dea et al. (2018)	Case control	ADHD: 8, Control: 7	Not mentioned	Medication naïve.	6–10 years	Electroencephalograph(EEG) recordings. Limitations: Small sample size.	To evaluate the differences between ADHD and healthy children of the power spectral values in delta, theta, alpha, beta and gamma bands, before, during and after sleep spindles.	Results show significant differences concentrated in the period immediately after spindle epochs, in the left hemisphere of the brain, in almost all bands, with greater values in control than in ADHD children (Left Anterior region- $p = .03$; Left central region – $p = .01$; Left posterior region- $p = .04$ for the Total values of Delta, Theta, Alpha, Beta & Sigma waves).
Saito et al. (2019)	Case control	ADHD: 21, Control: 18	100%/0%	Sleep inducing medication administered to all ADHD and 2 typically functioning participants.	5–13 years	Raven's Colored Progressive Matrices (RCPM), Das-Naglieri Cognitive Assessment System (DN-CAS), Mogras test (NoruPro Light Systems, Inc., Tokyo, Japan), Continuous Performance Test (CPT), EEG recordings and power spectral analysis. Limitations: No girls in sample, EEG frequency analysis based on one 1-hour recording, frequency analysis in stage 2 may be different from that of all-night recording, medication induced-sleep group included only few individuals.	To evaluate the power of slow sleep spindles during sleep stage 2 to clarify their relationship with executive function, especially attention, in children with ADHD.	Twelve-hertz frontal spindle EEG activity may have positive associations with sustained attention function (reaction time variability in CPT) ($r = .368$, $p = .0242$).
Ringli et al. (2013)	Case control	ADHD: 9, Control: 9	88%/12%	Within ADHD group: 6 medication naïve, 1 pretreated with MPH, 2 on MPH.	6–12 years	EEG recordings and Power spectral analysis. Limitations: Cross-sectional data with limited age range, which does not offer investigation of intraindividual cortical development or electrical neuronal activity, small sample size, effect of medication not examined.	To compare the sleep EEG of children with ADHD with healthy controls to explore differences in sleep SWA (Slow Wave Activity).	Children with ADHD showed a less mature topographical SWA distribution in comparison to healthy children of the same age and sex. (Calculated Power maps for each group showed that ADHD children exhibited 17% ($\pm$6% SE, $p < .01$) more SWA in a cluster of six central electrodes).

(continued)

Table 3. (continued)

Authors/ publication year	Study design	Participant characteristics: Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age	Primary measures, limitations and biases	Objective	Summary of main results: Interventions/comparisons/ outcomes
Miano et al. (2016)	Case control	ADHD: 30, Control: 25	70%/30%	Medication naïve.	8–13 years	K-SADS-PL, NEPSY-II, WISC-V, nocturnal video PSG, actigraphy, Multiple sleep latency test, CSHQ. Limitations: Small sample size and not comparable number of female participants.	Full sleep assessment using objective measures for ADHD and control children.	All ADHD children reported a history of sleep problems and slept less than 9 hour per night, indicating chronic sleep deprivation that should be evaluated as a possible unifying marker of ADHD (TST minutes, ADHD- 421.49 (±58.93) vs. Control- 462.8 (±69.57) $p < .022$).
Miano et al. (2019)	Case control	ADHD: 30, Control: 25	70%/30%	Medication naïve.	7.5–13.5 years	K-SADS-PL, NEPSY-II, WISC-V, nocturnal video PSG, actigraphy, Multiple sleep latency test. Limitations: Controls did not undergo actigraphic and MSLT recordings, all children underwent only one PSG recording (first night effect not controlled) andactigraphic recording was performed 1 week before PSG and MSLT, or the week after, depending on the needs of the child, without checking for naps before PSG recording.	To perform a detailed subjective and objective sleep investigation among ADHD children and controls.	Results confirm static and dynamic changes in SWA behavior in children with ADHD, which may reflect a maturational delay occurring at a vulnerable age, as a consequence of chronic sleep deprivation (In early sleep ADHD children showed a greater amount of delta power over the centro–parieto–occipital regions (Permutation test, FDR-corrected $p < .01$) In late sleep ADHD children, a greater amount of delta power over the centro–parietal regions were shown (Permutation test, FDR corrected, $p < .01$).
Příhodová et al. (2012)	Case control	ADHD: 14, Control:12	85%/15%	Medication naïve.	7–12 years	Nocturnal video PSG. Limitations: Scoring of sleep microstructure parameters (arousals) may differ from center to center, small sample size, more male participants.	To evaluate the microstructure of sleep in children with ADHD.	Sleep microstructure analysis using CAP revealed no significant differences between the ADHD group and the controls in any of the parameters under study.
Galland et al. (2010)	Case control	ADHD:27, Control:27	76%/24%	Randomly assigned 48-hour on-off medication protocol was used for ADHD children.	6–12 years	PSG, Behavioral Assessment Scales for Children (BASC), Sleep questionnaire completed by parents. Limitations: Small sample size, majority male participants, sleep debt incurred for drug use may have caused a rebound effect during washout period, so sleep duration and sleep latency compared on-off medication night could purely be rebound, dosages and formulations of MPH were not standardized, only one PSG recording was conducted for each condition (on-off medication), so information collected could be subject to a first-night effect.	To assess the effects of regular use of methylphenidate medication in children diagnosed with attention deficit hyperactivity disorder (ADHD) on sleep timing, duration and sleep architecture.	Methylphenidate reduces sleep quantity but does not alter sleep architecture in children diagnosed with ADHD (ADHD children had a significantly longer sleep latency on the medication night (65 vs. 38 minutes, $p = .002$), woke earlier in the morning (6:20 am vs. 6:45 am, $p = .03$), with a significantly shorter sleep period time (7.9 hours vs. 9 hours, $p < .001$) and had reduced sleep efficiency (81.7 vs. 87.3, $p < .001$); these medications effects held strongly when control data were taken into account).

(continued)

Table 3. (continued)

Authors/ publication year	Study design	Participant characteristics				Primary measures, limitations and biases	Objective	Summary of main results
		Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age			Interventions/comparisons/ outcomes
Galland et al. (2011)	Case control	ADHD: 28, Control: 28	76%/24%	Randomly assigned 48-hour on-off medication protocol was used for ADHD children.	6–12years	Nocturnal PSG, Sleep and breathing questionnaire. Limitations: Small sample size, majority male participants, parents were not blinded to study/medication status, snoring data extracted from shortened sleep times on the medication night in children with ADHD, contamination of the PSG data by "first night" effects.	To measure apnea-hypopnea indices and snoring in children diagnosed with ADHD and typically developing controls.	PSG data revealed no significant differences between control group and ADHD children for presence of an AHI >1 or snoring.
Prihodova et al. (2010)	Case control	ADHD: 31, Control :26	83%/17%	Not currently medicated.	6–12years	PSG (2 nights) and MSLT, CBCL and Conner's Parents rating scale. Limitations: More male participants, ADHD group had a homogenous make up with more combined subtype.	To evaluate the microstructure of sleep in children with ADHD.	No REM /Sleep function changes noted in ADHD.
Henriques Filho (2016)	Cross sectional	ADHD: 61	57%/43%	Temporary discontinuation of psycho-stimulant medication before assessment.	8–13years	PSG and P300 evoked potential test. Limitations: The use of subjectively reported sleep parameters alongside the objective measures, would have given a more comprehensive result, keeping in mind the environmental-social-cultural variables at play.	To determine prevalence of OSA in a group of children with ADHD and to compare amplitude and latency of the P300 potential between ADHD children with and without associated OSA.	Significant decreased amplitude of the P300 potential was observed in children with OSA+ADHD when compared with children with only ADHD (without OSA) ($F=3.661$, $p=.028$). The results were confirmed by Spearman correlation analysis, which showed a significant correlation between increased AHI and decreased P300 amplitude (test 1: $r=-.631$, $p=.0001$; test 2: $r=-.672$, $p=.0001$; test 3: $r=.651$. $p=.0001$) in the ADHD+ OSA group).
*Wiebe et al. (2013)	Case control	ADHD: 26, Control: 56	62%/ 38%	4 children with ADHD on medication (washout period of 48hours before sleep assessment).	7–11years	PSG, actigraphy, Multiple sleep latency test, Epworth Sleepiness Scale. Limitations: Small sample size, paediatric norms not well established for the MSLT, restless legs syndrome was not measured.	To assess the association between habitual sleep patterns and one night of PSG measured sleep with daytime sleepiness in children with ADHD and typically developing children.	Actigraphy measures of sleep restlessness (time awake ($r=.48$) and activity ($r=.48$) during the night), as well as time in slow-wave sleep ($r=.46$), were positively related to mean sleep latency on the multiple sleep latency test in children with ADHD (@ $p<.05$).

PSG has also been used to assess other sleep features in children with ADHD. Multiple Sleep Latency test (MSLT) using PSG measures among 7 to 11-year-old children with ADHD revealed that longer time in slow-wave sleep were positively related to mean sleep latency and longer actigraph-measured time awake and more actigraph-measured activity were associated with longer mean latency on the MSLT (Wiebe et al., 2013). In another MSLT study, although mean sleep latency showed no inter-group differences between the ADHD ($n=31$) and control group ($n=26$), for the ADHD group a significant inter-test variability was observed, that is sleep latency values displayed statistically significant differences between some of the tests during the day (between the 1st and the 2nd,4th and 5th test) (Prihodova et al., 2010). Miano et al. (2019) report that children with ADHD ($n=30$, 8–12 years) showed a higher Apnea-Hypoapnea Index (AHI) than control group children ($n=25$). The particular functional importance of sleep-disordered breathing in children with ADHD was shown in a study in which 61 (8–13 year old) children with ADHD with or without obstructive sleep apnea underwent an oddball auditory attention test coupled with detection of P300 (Event related potentials) followed by an all-night PSG, and revealed significant decreased amplitude of the P300 potential in children with Obstructive sleep apnea (OSA) +ADHD, when compared with children with only ADHD (Henriques Filho, 2016). However, there is not complete concordance between studies in this area: a two night PSG data (6–12 years) revealed no significant differences between control group ($n=28$) and children with ADHD ($n=28$) for presence of an AHI >1 (where a threshold of >1 AHI event per hour is of clinical relevance) or snoring (Galland et al., 2011). Further, within the same age group, Prihodova et al. (2010) found no significant difference in the occurrence of sleep disordered breathing between ADHD ($n=31$) and control children ($n=26$) following a 2 night PSG and MSLT study. With regards to periodic limb movements recorded during PSG, Akinci et al. (2015) found that the periodic limb movement index (PLMI) was higher in children with ADHD ($n=28$) compared to the control group ($n=15$). Prihodova et al. (2010) found no significant difference in the occurrence of periodic limb movements in sleep between 6 and 12-year-old ADHD ($n=31$) and control ($n=26$) children following a 2 night PSG and MSLT study, although a statistically significant difference was found in the trend for periodic limb movement index (PLMI) between two nights (a decrease of PLMI in the ADHD group and an increase of PLMI in the control group during the second night).

Circadian Rhythms and Chronotype in Attention Deficit Hyperactivity Disorder

Cortisol rhythms. A number of studies have examined diurnal variations in salivary measures for cortisol, which is under strong circadian control as the end product of the hypothalamic-pituitary-adrenal (HPA) axis in children with ADHD and matched control groups of typically functioning children (studies detailed in Table 4). Salivary Cortisol awakening response (CAR) was found to be lower in children with ADHD ($n=62$) than in controls ($n=40$) with a pronounced difference for the ADHD combined presentation (hyperactive and inattentive; Angeli et al., 2018). Contrary to the above finding, a couple of studies revealed no difference in CAR response between ADHD and control groups (Imeraj et al., 2012; Buske-Kirschbaum et al., 2019). Children with ADHD treated with Methylphenidate and with higher morning cortisol levels were found to have better CPT performance (specifically for impulse control) compared with those with lower levels of the same ($n=50$, 6–12 years; Wang et al., 2017). For diurnal cortisol rhythms, children with ADHD compared to typically developing children had significantly lower levels of salivary cortisol at a number of time points during the day (Angeli et al., 2018). Interestingly, Imeraj et al. (2012) showed that for cortisol measures at a number of different time points in a day (waking, noon, 4pm and 8pm) orthogonal contrasts of the overall ADHD group ($n=33$, 6–12 years) and the control group ($n=33$) did not reveal any group differences, although orthogonal contrasts comparing the ADHD + Oppositional defiant disorder and the ADHD groups revealed significant difference in cortisol measures. In this study specifically, ADHD + ODD subgroup showed significantly higher morning and lower evening levels, resulting in a steeper linear decrease in cortisol levels throughout the day as compared to the ADHD subgroup (Imeraj et al., 2012). As a measure of total cortisol output, lower levels of AUCg (area under the curve with respect to ground) and AUCi (area under the curve with respect to increase) for "wake to bed" period was found in 6 to 10-year-old children with ADHD combined presentation and ADHD-I, suggesting lower overall cortisol secretion over a 24-hour period (Angeli et al., 2018). On the other hand, Buske-Kirschbaum et al. (2019) demonstrated no group differences for AUCg diurnal profiles among ADHD ($n=34$, 6–12 years) and control group children.

For stress responses in salivary cortisol, Angeli et al. (2018) showed no statistically significant differences in pre- and post-stress responses (induced through venipuncture) between the ADHD ($n=62$) and control group ($n=40$). Additionally, these authors did not report different diurnal profiles for the enzyme alpha-amylase for children with ADHD, although after stress response there was an increase in alpha-amylase in the control group but not the ADHD-combined and inattentive groups (Angeli et al., 2018). Buske-Kirschbaum et al. (2019) measured stimulated cortisol secretion (via acute psychosocial stressor elicited by the Trier Social Stress Test), a less pronounced cortisol response was found in the ADHD group ($n=34$, 6–12 years) compared to

Table 4. Summary of Eleven Included Studies Focusing on Circadian Rhythm Markers Among ADHD Children.

		Participant characteristics						Summary of main results
Authors/ publication year	Study design	Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age	Primary measures, limitations and biases	Objective	Interventions/comparisons/outcomes
Angeli et al. (2018)	Case control	ADHD: 62, Control: 40	68%/32%	Not currently medicated.	6–10 years	Assessment of salivary samples, Raven's progressive matrices and K-SADS. Limitations: Small sample, diurnal variations assessed for one day, the exact time point of awakening and bedtime was not recorded, stress imposed by venipuncture was not very effective in eliciting stress response of both limbs (stress system and HPA axis).	To assess diurnal rhythms and stress responses in children with ADHD, via consecutive measurements of specific, extensively used, salivary biomarkers (cortisol for the HPA axis and α- amylase for SNS).	Results revealed that children with ADHD-C had lower mean cortisol values both 30 minutes after awakening (ADHD-C-14.60 (4.43) vs. No ADHD-19.38 (7.10) $p = .002$) and at 18:00 hour than controls (ADHD-C-7.16 (1.93) vs. No ADHD-9.01 (4.18) $p = .018$), as well as lower mean Cortisol Awakening Response (CAR) (ADHD-C-−.40 (4.73), ADHD-I-.56 (8.26) & No ADHD-4.50 (3.97) $p = .001$) and Area Under the Curve for "wake to bed" period (AUCi) values of cortisol (ADHD-C- 1775.88 (693.26), ADHD-I-1868.12 (621.66), No ADHD- 2804.98 (1707.00) $p = .001$), mean CAR and cortisol AUCi were lower in children with ADHD-I than the control group.
Imeraj et al. (2012)	Case control	ADHD: 33, Control:33	82%/18%	28 children took MPH (washout period of 72 hours prior to testing)	6–12 years	Salivary cortisol analysis, CBCL, Level of puberty achieved, BMI, Sleep and wake time (weekend and weekday). Limitations: salivary cortisol collected in naturalistic environment resulted in some noncompliance, overall small sample, leading to small sample sizes of clinical subgroups (ADHD + ODD and ADHD), subjects with ODD without ADHD not included, activity levels not controlled, no objective test of arousal (i.e., multiple sleep latency test).	To examine across-the-day cortisol variations in ADHD and control children.	Longitudinal analyses to evaluate cortisol profiles across the day revealed a significant Group · Time effect ($p < .001$), the ADHD subgroup showed a flatter slope with relative morning hypo-arousal and evening hyper arousal, whereas the ADHD + ODD subgroup showed a steeper slope with relative morning hyperarousal and evening hypo-arousal ($p < .001$). Control vs. ADHD (overall) group difference in noon Cortisol levels (showing a large effect size of 0/83 and ADHD + ODD versus ADHD group difference in the evening Cortisol levels (showing a large effect size of .91) however for 4 pm effect size is .25.
Buske-Kirschbaum et al. (2019)	Case control	ADHD: 34. Remaining sample: 111 (Atopic Eczema- 42, ADHD +Atopic Eczema- 31, controls- 47)	81%/9%	All ADHD children were medication free for more than 48 hours prior to testing and all Atopic Eczema patients were anti-inflammatory medication free for 4 weeks.	6–12 years	Psychological stress test (TSST-C), SCORAD, Salivary cortisol, FBB-ADHS questionnaire. Limitations: Small sample size, participants with AE to (mild to mild cases, ethics committee requirement), low symptom severity might hindered detection of alterations in HPA axis function that would be visible with pronounced AE symptomatology, changes in sleep quantity and quality not assessed, cross-sectional investigation provides only correlational data.	To evaluate HPA axis function, salivary cortisol in response to psychosocial stress (Trier Social Stress Test for Children, TSST-C), after awakening (cortisol awakening response, CAR), and throughout the day (short diurnal profile) and hair cortisol capturing long-term HPA axis activity were assessed.	No alteration of the cortisol response to the TSST-C was observed in children with AE, however, in children with AE, increased ADHD-like behavior was associated with a reduced HPA axis response to acute stress (for children with AE (with or without an additional diagnosis of ADHD), Impulsivity, Inattention, and Total scores were negatively correlated with the percent change in salivary cortisol in response to the TSST-C (effect sizes $r = -.21$, $p = .031$, $r = -.29$, $p = .004$, and $r = -.23$, $p = .020$, respectively). Groups did not differ in CAR, short diurnal profile, and hair cortisol.

(continued)

Table 4. (continued)

Authors/ publication year	Study design	Participant characteristics: Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age	Primary measures, limitations and biases	Objective	Summary of main results: Interventions/comparisons/outcomes
Wang et al. (2017)	Case control	ADHD: 50, Control: 50	80%/20%	Not currently medicated.	6–12 years	SNAP-IV, CBCL, CPT, ADHD-RT, Saliva Cortisol test. Limitations: Saliva samples collected from patients in hospital but from healthy controls in school, no measurements of cortisol increment levels were made after baseline and during first month of treatment, waking time of participants was not precisely identified, patients with ODD or conduct disorder excluded.	To determine the trend in cortisol levels in children with ADHD and controls.	The cortisol levels of ADHD patients increased significantly after 1 month of MPH treatment (significantly higher than those at pre-treatment; mean difference = .11, $p = .046$). before decreasing to an intermediate level, but were significantly positively correlated with neuropsychological performance (salivary Cortisol was found to be independently and significantly correlated with impulse control ($\beta = -.006$, $p = .003$).
Imeraj et al. (2011)	Case control	ADHD: 30, Control: 30	80%/20%	Not currently medicated.	6–11 years	Actigraph, Actiheart, CBCL, Demographic variables. Limitations: higher stress levels expected to elevate heart rate levels, so that differential results in ADHD may be due to secondary conditions rather than to ADHD itself, pubertal stage/ sex/stimulant medication/caffeine use/presence of nightmares/regular physical training.	To investigate 24-hour heart rate patterns under natural environmental conditions among ADHD and control children.	Heart rate levels were overall higher in the ADHD group ($p < .01$)—with the largest effects during afternoon (effect size- .69) and night (effect size – .57) ($p < .001$)—in a model controlling for age and children with ADHD showed higher activity levels during daytime (especially early afternoon) (effect size – .83) ($p < .05$), but not during night-time (effect size- .11).
Langevin and Ramdé (2012)	Case control	ADHD: 10, Control: 5	80%/20%	5 children with ADHD on psychostimulant medication.	7–9 years	Sleep log, Actimetry, French version (SWAN-F). Limitations: All data from Actiware not assessed, time of study coincided with week before Christmas holidays, leading to differences in excitement level of children (particularly ADHD), small sample size.	To verify that the shortening photoperiods of winter contribute to increasing the nocturnal and diurnal agitation of children with ADHD and that lengthening photoperiods diminish it.	Results show a significant baseline difference between the nocturnal motor movements of the ADHD children (on and off psychostimulants) and those of the control children ($p = .008$) during the winter peak (December). The same was also true for diurnal agitation (inattention, hyperactivity) between the ADHD (not on psychostimulants) and control group ($p = .008$) for both the winter (December) and summer (June) time points.
Novakova et al. (2011)	Case control	ADHD: 34, Control: 43	70%/30%	Not currently medicated.	6–12 years	Saliva specimens were collected in four different sessions around the time of the spring and autumn equinoxes when the natural light lasted 11.2 ± .9 hour. Limitations: Small sample, majority male participants.	To examine the salivary Melatonin rhythms among children with ADHD and controls.	Age based categorization of children, revealed significant differences between the ADHD and control group in the melatonin rhythm waveform (when the profiles of the 6–7-year-old and 10–12 year-old ADHD and control children were compared, significant differences in melatonin values between these ages at 22:00 hour ($p < .001$) and 06:00 hour ($p = .001$), suggesting that the entire profile in the older children was phase delayed relative to the younger children.

(continued)

Table 4. (continued)

Authors/ publication year	Study design	Participant characteristics				Primary measures, limitations and biases	Objective	Summary of main results
		Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age			Interventions/comparisons/outcomes
Paclt et al. (2011)	Case control	ADHD: 34, Control: 43, Anxiety: 11	Not mentioned	Not currently medicated.	6–12 years	Saliva specimens were collected in four different sessions during the school year, around the time of the spring and autumn equinox, when the natural light lasted 11.2 hour ± .9 hour. Limitations: Small sample size.	To examine salivary Melatonin levels during Spring and Autumn equinoxes among ADHD, controls and Anxious children.	More symptoms of conduct disorder elevated positive or negative correlations between psychopathology and saliva level of melatonin in ADHD and anxiety samples (for example ADHD children exhibit positive correlation ($p < .01$), correlation of .438 between the melatonin level at 18 hours and hyperactivity score at Conner's scale and correlation ($p < .01$), .541 for conduct disorder score in Conner's scale).
Tarakçioğlu et al. (2018)	Case control	ADHD: 53, Control: 38	81%/19%	Not currently medicated.	6–12 years	CSHQ, Childhood Chronotype Questionnaire (CCTQ). Limitations: small sample size, Turkish version of CCTQ not enough sensitivity and specificity to assess preferences of morningness and eveningness, in both groups, almost all parents reported eveningness type, only subjectively assessed parental reports used, comorbid psychiatric concerns not analyzed statistically due to inadequate number of comorbid cases.	To investigate the relationship between circadian characteristics and behavioral problems in children with ADHD and controls.	ADHD children had more sleep-onset problems (Bedtime resistance- ADHD: 12.5 (2.5) vs. Control: 11 (3.2) @ $p < .023$) and parasomnias – ADHD: 9.5 (2) vs. Control: 8.2 (1.5) @ $p < .003$) compared to healthy controls and circadian preferences did not differ between the groups in CCTQ scores.
Van der Heijden et al. (2018)	Case control	ADHD:12 (within psychiatric group: 112), Control: 271	70%/30%	16% of children with ADHD were on Melatonin and 79% of children with ADHD were on MPH.	6–12 years	CCTQ, CSHQ, Childhood Sleep Disturbance Scale (CSDS), CBCL (Child Behavior Checklist), Parent questionnaire for Sleep onset latency (SOL), Electronic Media use rating scale. Limitations: Assessments based on subjective parent reports (use of objective measures ex. actigraphy or dim light melatonin onset would have strengthened results), for etiological role of sleep hygiene and chronotype long term cohort studies required, influence of medication.	To understand the association of sleep patterns with sleep hygiene among children with ADHD, ASD and typically developing (TD) controls.	Chronotype did not differ significantly between groups, but evening types were associated with sleep problems in ADHD ($\beta = .471$, $p = .002$) and TD ($\beta = .317$, $p < .001$) and more sleep problems were shown in ADHD than TD (Disorders of initiating and maintaining sleep @ $p < .001$, sleep-wake transition disorders @ $p < .001$, disorders of excessive somnolence @ $p < .001$, sleep hyperhidrosis@ $p < .01$, total sleep problem score @ $p < .001$).
Schwichtenberg et al. (2016)	Longitudinal	99	53%/47%	Not currently medicated.	2–6 years	Infant prematurity and medical risk records, CBCL, Conner's questionnaires, Actigraph (micromini motion logger), Abbreviated Battery IQ (ABIQ), Illness related health care visit records. Limitations: TSA framework did not provide a model of daytime napping or a midday drop in activity, large and non-random attrition from 3 to 6 years (mostly non-caucasian), thereby limiting generalizibility, bidirectional nature of sleep and developmental/health concerns not focused on.	To assess the associations between toddler circadian sleep/activity patterns and later developmental, behavioral, attentional, and health concerns in this at-risk population.	Toddlers with patterns that closely aligned with the specified 24-hour circadian cycle (SCC) had higher abbreviated intelligence quotient scores at 3 years of age (beta coefficient of .235 for the closest fit to prototypical circadian pattern and cognitive skills (ABIQ scores) at age 3 @ $p < .05$), at 6 years had lower risk for illness-related medical visits (incidence rate ratio (IRR) of .749 for the closest fit to prototypical circadian pattern and child illness related health care (total medical attention) at age 6 @ $p < .001$).

the control group, along with a negative correlation between ADHD symptom score and cortisol stress response (higher the ADHD symptoms, lower the cortisol response).

Cardiovascular rhythms. When considering diurnal profiles for heart rate and activity patterns in children with ADHD, Imeraj et al. (2011) found that heart rate levels were significantly higher in the ADHD group ($n=30$) compared to the control group ($n=30$), with these group difference particularly expressed during nighttime and afternoon. In the same study, 24-hour activity patterns measured through actigraphy revealed that between 9 and 10 a.m. and between 11 a.m. and 4 p.m. activity levels were significantly higher in the ADHD group (Imeraj et al., 2011). On similar lines, when seasonal changes in activity profiles were investigated (with two time-point measurements, T1: December & T2: June) difference between the children with ADHD's levels ($n=10$) of nocturnal motor movement in December and in June was greater than that of the control group children ($n=5$; Langevin & Ramdé, 2012). In this study, it was found that children with ADHD treated with psychostimulants had higher level of nocturnal motor movements when compared to untreated children with ADHD. With regard to seasonal change in sleep, children with ADHD had significant improvement in sleep quality in T2 as compared to controls.

Melatonin, chronotype, and diurnal activity rhythms. When assessing melatonin rhythms, a study showed that although children with ADHD did not differ from control children in melatonin peaks and maximum night-time levels, their 24-hour melatonin profiles differed significantly, with the duration of the nocturnal melatonin signal shortened in children with ADHD between the ages of 10 and 12 years, due to the significantly earlier morning melatonin decline in the ADHD group ($n=34$; Novakova et al., 2011). In another study by Paclt et al. (2011) found no difference in melatonin levels between the 6 to 12-year-old ADHD ($n=4$) and control group ($n=43$). Tarakçioğlu et al. (2018) investigated chronotype preference among children with ADHD ($n=53$) and typically developing ($n=38$) children aged 6 to 12 years using the Children's chronotype questionnaire, and revealed no specific morning/evening preference for the ADHD group compared to controls. Van der Heijden et al. (2018) similarly found that chronotype did not differ significantly between groups (ADHD, control and ASD), but evening type was associated with sleep problems in children in the ADHD ($n=44$) and TD groups ($n=243$). When sleep/activity data was collected at 2 years via actigraphy for 99 preterm children, and the results were assessed using time series analysis by comparing each child's sleep/activity circadian cycle, it was found that higher toddler activity level was associated with fewer teacher-reported ADHD symptoms and a lower risk for illness-related medical visits at 6 years (Schwichtenberg et al., 2016).

Functional Consequences of Sleep Functioning in Children with ADHD

Behavioral consequences. Emotional and behavioral problems may contribute to sleep problems in ADHD, and may also exacerbate negative impacts of disturbed sleep in children with ADHD (relevant studies detailed in Table 5). It is reported that anxiety ratings in children aged 7 to 13 years with ADHD ($n=181$) were associated with bedtime resistance and sleep anxiety, whereas depressive symptoms were associated with shorter sleep duration and increased day time sleepiness, along with a unique association of oppositional defiant symptoms with shorter sleep duration; further girls with ADHD had more sleep problems and greater anxiety than boys (Becker, Froehlich, et al., 2016). As such, anxiety and/or depression exacerbated total sleep problems and further interacted with ADHD symptoms to predict sleep length and sleep duration problems (Tong et al., 2018, ADHD symptoms $n=82$, 9–12 years). Similarly, teacher reported daytime sleepiness in 5 to 13-year-old children with ADHD was associated with higher levels of emotional and behavioral problems in the classroom ($n=257$, Lucas et al., 2019). Poorer caregiver reported well-being (behavioral and emotional problems) in similarly aged children with ADHD ($n=186$) was associated with transient or persistent sleep problems (Lycett et al., 2016). Further, sleep problems in ADHD are positively correlated with parent-reported perfectionism, psychosomatic complaints and anxious thoughts (Blunden et al., 2011, $n=88$) and predicted adolescent irritability (Mulraney et al., 2017, $n=140$). Children with ADHD and internalizing comorbidities had higher reported sleep anxiety than children with ADHD alone (Lycett, Sciberras, et al., 2015, $n=392$). Mulraney et al. (2016) found that although there is a weak evidence of a bidirectional relationship between sleep problems and emotional problems in 270 children with ADHD (stable over a 12-month period among age of 5–13 years), they did not appear to influence each other after the 12-month period.

A longitudinal study ($n=3,800$) exploring the relationship between sleep and the presence of pre-teen delinquency in relation to ADHD symptomatology and diagnosis revealed that sleep problems during childhood were significantly associated with higher odds of ADHD symptomatology, receiving a ADHD diagnosis and pre-teen delinquency (ORs of 2.38–8.10; Jackson & Vaughn, 2017). However, the association between sleep problems and pre-teen delinquency was no longer significant once ADHD symptomatology and diagnosis were considered (Jackson & Vaughn, 2017). In a study exploring the effects of circadian and behavioral tendencies on sleep onset problems for children with ADHD, it was found that for both the 7 to 11 years aged clinical ($n=26$) and control ($n=49$) group participants' externalizing problems yielded significant independent

Table 5. Summary of Thirty Four Included Studies Focusing on Consequences of Sleep Functioning in Children with ADHD.

		Participant characteristics						Summary of main results
Authors/ publication year	Study design	Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age	Primary measures, limitations and biases	Objective	Interventions/comparisons/outcomes
Becker et al. (2018)	Cross sectional	ADHD: 181	86%/14%	Not currently medicated.	7–13 years	CSHQ, Vanderbilt ADHD Diagnostic Parent Rating Scale (VADPRS), Revised Child Anxiety and Depression Scales, Parent Version (RCADS-P). Limitations: Cross-sectional study cannot indicate causality, few children in sample met criteria for comorbid diagnoses thereby limiting generalizability, sole reliance on parent-reported rating scales which may have contributed to mono-informant biases.	To examine gender based differences in sleep functioning among ADHD children.	Specific ADHD subtypes associated with specific sleep concerns (for example sleep onset delay $r = .15$ @ $p < .05$ level; sleep duration $r = .17$ @ $p < .01$ level; night wakenings $r = .24$ @ $p < .01$ level and parasomnias $r = .26$ @ $p < .001$ level associated with ADHD- Hyperactivity/ Impulsivity) and girls had poorer sleep functioning than boys across most sleep functioning domains (75% of girls ($n = 42$) met the established cut-off for having sleep problems, compared to 53% of boys ($n = 66$), total sleep disturbance, sex based difference Effect Size: Cohen's $d = .58$)).
Lucas et al. (2019)	Cross sectional	ADHD: 257	74%/26%	201 children with ADHD were on stimulant medication.	5–13 years	CSHQ, Teacher's Daytime Sleepiness Questionnaire, Strength and Difficulties Questionnaire (SDQ). Limitations: cross-sectional design (no focus on additional predictive factors, beyond target variables), average study sample were taking ADHD medication; thus not clear whether results would generalize to less impaired sample of patients.	To determine if sleep problems and daytime sleepiness were associated with the social, emotional, and behavioral school-based functioning of children with ADHD.	CSHQ subscale night waking was correlated with teacher-rated daytime sleepiness ($r = .25$ @ $p < .001$) and this in turn was associated with higher levels of emotional ($\beta = .36$; 95% CI = .24–.49, R squared = .25) and behavioral problems ($\beta = .51$; 95% CI = .40–.62, R squared = .37).
Lycett et al. (2016)	Cross sectional	ADHD: 186	86%/14%	143 children with ADHD were on medication (short and long acting MPH, Dexamphetamine, Atomoxetine).	5–13 years	CSHQ, Paediatric Quality of Life Inventory. Limitations: Child/family well-being already markedly worse for children experiencing transient/persistent sleep problems, study could further explore long term follow up format to delineate this relationship.	To examine the longitudinal relationship between sleep problem trajectories and well-being in children with ADHD.	Children with either transient (95% confidence interval (CI) .4, 1.0; $p < .001$) or persistent sleep problems (95% CI, .2–1.2; $p = .004$) had greater (7 standard deviation units higher) caregiver-reported conduct and emotional problems over the 12-month period.
Blunden et al. (2011)	Cross sectional	ADHD: 88	78%/22%	Not currently medicated.	6–13 years	Victorian Cancer Council food frequency questionnaire (FFQ), Sleep Disturbance Scale for Children (SDSC). Limitations: No measure for preservatives and additives contained in foods/ drinks, no control group and no randomized design (difficult to ascertain any causal pathway), self-selected sample, sleep-dietary intake subjectively assessed by parental reports, psychosocial factors not measured.	To investigate relationships between sleep and dietary macronutrient intake in children with ADHD.	Parents who reported more sleep disturbance also reported a higher intake of carbohydrate: ($r = .26$, $p < .05$), poly fats ($r = .25$, $p < .05$), and, most particularly, sugar ($r = .30$, $p < .01$) which was also a significant predictor of night time sweating (beta coefficient of .261, $p = .018$, R Squared = .22).

(continued)

Table 5. (continued)

Authors/ publication year	Study design	Participant characteristics: Sample size and diagnosis	Participant characteristics: Gender (male%/ female%)	Participant characteristics: Medication use	Participant characteristics: Age	Primary measures, limitations and biases	Objective	Summary of main results: Interventions/comparisons/outcomes
Mulraney et al. (2017)	Longitudinal study	ADHD:140	89%/11%	139 children with ADHD were on medication.	5–13 years	Disorders of initiating and maintaining sleep (DIMS) subscale from the Sleep Disturbance Scale for Children (SDSC), Strengths and Difficulty questionnaire, ADHD-RS-IV, Affective Reactivity Index (ARI), Depression Anxiety Stress Scale (DASS-21) for parents, Socio-Economic Indexes for Areas (SEIFA) Disadvantage Index. Limitations: no assessment of irritability at baseline, 35% of original sample participated in follow-up which may limit the generalizability of results, only 15 girls in sample (results cannot be generalized to girls).	To study if internalizing and externalizing problems predict adolescent irritability 3 years later and if child (behavior, sleep, school attendance) and parent factors (parental mental health) are associated with adolescent irritability.	Irritability was associated with increased attention-deficit/ hyperactivity disorder symptom severity (parent-report ($\beta = .45$; 95% CI = [.30, –.60]; $p < .001$) and teacher-report ($\beta = .33$; 95% CI = [.16, –.49]; $p < .001$) and sleep problems ($\beta = .28$; 95% CI = [.12, –.45]; $p = .001$); poorer emotional ($\beta = .47$; 95% CI = [.30, –.64]; $p < .001$), behavioral ($\beta = .64$; 95% CI = [.54, –.74]; $p < .001$) and social functioning ($\beta = .44$; 95% CI = [.28, –.61]; $p < .001$); and poorer parent mental health ($\beta = .33$; 95% CI = [.22, –.53]; $p < .001$, stress level).
*Lycett, Sciberras, et al. (2015)	Cross sectional	ADHD: 392	85%/15%	333 children with ADHD were on medication (Psycho stimulants and Atomoxetine)	5–13 years	7-day sleep log, CSHQ, ADHD-RS-IV, sleep problems severity assessment, Depression anxiety stress scale, Anxiety Disorders Schedule for Children-IV, Socio-Economic Indexes for Areas. Limitations: All sleep measure's reliance on parental report reflect common effect of parental perception across the measures, findings limited to behavioral sleep problems, two-thirds of parents returned the sleep log, which may have under- or over-ascertained problem sleepers.	To examine the association between sleep problems and internalizing and externalizing comorbidities in children with ADHD.	Compared to children without comorbidities, children with co-occurring internalizing and externalizing comorbidities were more likely to have moderate/ severe sleep problems (adjusted OR 2.4, 95% CI 1.2; 4.5, $p = .009$) and problematic sleep across six of seven sleep domains (bedtime resistance, Sleep duration, parasomnias, night waking, daytime sleepiness, sleep anxiety and total sleep problems).
Mulraney et al. (2016)	Cohort study	ADHD:270	85%/15%	214 children with ADHD were on medication (details not mentioned)	5–13 years	Strengths and Difficulty questionnaire, ADHD-RS-IV, CSHQ. Limitations: Reliance on caregiver report (sleep and internalizing/externalizing problems), CSHQ does not adequately capture sleep-onset latency or sleep quality which objective measures (eg, actigraphy) might ideally assess, study design does not examine longitudinal relationship between variables.	To investigate the bidirectional relationship between sleep problems and internalizing/ externalizing problems.	Sleep problems at baseline predicted emotional problems at 6 months ($r = .17$, $p < .01$), and emotional problems at baseline predicted sleep problems at 6 months ($r = .07$, $p < .05$) and the relationship between these two variables was stable over time (12 months).

(continued)

Table 5. (continued)

Authors/ publication year	Study design	Participant characteristics				Primary measures, limitations and biases	Objective	Summary of main results
		Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age			Interventions/comparisons/outcomes
Gruber, Fontil, et al. (2012)	Case control	ADHD:26, Control:49	65%/35%	9 children with ADHD were on medication (MPH, Atomoxetine)	7–11 years	CBCL, CSHQ, PSG, Child morning-evening preference scale (CMEP), Child morning-evening preference scale (CMEP). Limitations: Participants below cut-off score on Pediatric Sleep Questionnaire (sleep disordered breathing not assessed objectively) objective circadian measures, ex. salivary melatonin/core body temperature not used, one-third ADHD children prescribed stimulants to control symptoms had potentially short wash-out periods (transient worsening of symptom might occurred after medication was stopped).	To determine the relative contributions of circadian preferences and behavioral problems to sleep onset problems in children with ADHD.	Externalizing problems yielded independent contributions to parental reports of bedtime resistance (beta coefficient of .90 for the CBCL externalizing T-score and bedtime resistance @ $p < .05$, R squared = .10), whereas an evening circadian tendency contributed to parental reports of sleep onset delay (beta coefficient of .37 for Eveningness -score and CSHQ sleep onset delay @ $p < .01$, R squared = .20) and PSG-measured sleep-onset latency (beta coefficient of 8.16 for Eveningness -score and PSG sleep onset delay @ $p < .01$, R squared = .16).
Thomas et al. (2018)	Cross sectional	ADHD: 392 (out of which ADHD+ASD:93, ADHD:299)	85%/15%	Within ADHD group 255 were on medication and within ADHD+ASD group 78 were on medication for ADHD symptoms.	5–13 years	Anxiety Disorders Interview Schedule of Children IV/Parent version (ADIS-C), CSHQ, Peer problem subscale of the Strengths and Difficulties Questionnaire (SDQ). Limitations: Reliance on parent report of ASD diagnosis, rather than clinical interview, reliance on parent report of behavioral sleep problems, no data for children taking medication for sleep problems, ex. melatonin, which could have influenced results.	To examine the types and severity of behavioral sleep problems experienced by children with comorbid ADHD + ASD compared with those with ADHD.	Children with ADHD + ASD experienced similar levels and types of behavioral sleep problems compared with those with ADHD (Effect sizes ranged from −.05 to .12, indicating very small differences between the two groups) and co-occurring internalizing and externalizing comorbidities was associated with sleep problems in this group ($\beta = .19$).
Soehner et al. (2019)	Longitudinal	ADHD: 145 (among, High risk youth:267 Community comparison control group:217)	50.6%/49.4%	56 participants from the entire group were on Stimulant/ non stimulant ADHD medication and 60 were on Antipsychotic, antidepressant, mood stabilizer and sedative/ hypnotic medication.	10–18 years	School Sleep Habits Survey (SSHS), KSADS-PL, Child Affective Lability Scale (CALS), Screen for Child Anxiety Related Emotional Disorders (SCARED), Disruptive Behavior Disorders Rating Scale. (DBDRS). Limitations: Identified longitudinal sleep-psychopathology associations, but causal interpretations cannot be made, missing data, sleep assessed via self-report; accurate report of sleep patterns can be affected by presence of psychiatric symptoms, objective, youth-report, and parent-report measures would be more accurate.	To identify sleep patterns that longitudinally change in conjunction with psychiatric symptom severity in at-risk youth.	Sleep predictors accounted for 33.1% of the explained variance (5.3% total variance) in the multivariate psychiatric symptom outcome.

(continued)

Table 5. (continued)

Authors/ publication year	Study design	Participant characteristics				Primary measures, limitations and biases	Objective	Summary of main results
		Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age			Interventions/comparisons/outcomes
*Moreau et al. (2013)	Cross sectional	ADHD:43	58.1%/41.9%	31 children with ADHD on medication (extended and immediate release psychostimulants, Atomoxetine and psychostimulants + Atomoxetine.	6–13 years	Conner's Continuous Performance Test (CPT), BRIEF (Behavior Rating Inventory of Executive Functions), Actigraph, Sleep diary, CSHQ, Conner's parent rating scale, K-SADS. Limitations: Small sample size, heterogenous in terms of medication use, half of sample had inattentive subtype only, due to cross-sectional nature, causality between sleep disturbances and daytime functioning measures cannot be inferred.	To investigate potential relationships between two measures of sleep impairments (i.e., sleep duration and sleep efficiency [SE]) and attention and executive functioning in children with ADHD.	Shorter sleep duration was associated with a range of executive functioning problems as reported by the parents ((Beta coefficient of .60 for total sleep time for behavioral regulation @ $p < .001$, R squared = .38 and Beta coefficient of .63 for total sleep time for global executive composite score @ $p < .001$, R squared = .29 from the BRIEF rating scale).
Kidwell et al. (2017)	Longitudinal	271 children	50.6%/49.4%	Not mentioned.	3–11 years	Sleep problems subscale of the Child Behavior Checklist (CBCL), CSHQ, Executive control battery, Conner's teacher's rating scale. Limitations: Sleep assessed at age 3 limited by parent-report format, ADHD symptoms may not be well-measured in 3-year-olds as in older children, ADHD symptoms assessed in community sample (teacher-report), sample primarily European American, which is regionally representative of the Midwest of United States.	To examine longitudinal associations among sleep, executive control, ADHD symptoms in children.	Sleep problems and executive control deficits early in development were associated with increased risk for ADHD symptoms in elementary school. Those with both sleep problems and EC deficits experienced a trend toward more inattention symptoms, ($b = -16.88$, SE = 8.89, $p = .058$). Those with both sleep problems and EC deficits experience more hyperactivity symptoms than those with high EC, ($b = -18.59$, SE = 9.11, $p = .041$).
Cremone, Kurdziel, et al. (2017)	Case control	ADHD:18, Control:15	72%/28%	2 children with ADHD were on medication (abstained from medication for 48 hours prior to testing)	4–8 years	PSG, Go/No-Go task to assess inhibitory control and sustained attention, Diagnostic Interview Schedule for children-IV. Limitations: Small sample size, more male participants.	To examine sleep microstructure in ADHD and typically developing children and its effect on executive functions.	Inhibitory control/sustained attention was not improved following overnight sleep in ADHD children although REM theta activity was greater in children with ADHD. Theta activity recorded during REM was significantly greater in the ADHD group compared to the TD group ($p = .014$). Region specific differences in theta activity between ADHD and controls showed greater activity in the frontal ($p = .038$) and central ($p = .081$) electrode locations for the ADHD group. Unlike, the control group, for ADHD group, neither scores for inhibitory control ($p = .38$) nor sustained attention ($p = .48$) changed compared to baseline after sleep in the morning.

(continued)

Table 5. (continued)

Authors/ publication year	Study design	Participant characteristics				Primary measures, limitations and biases	Objective	Summary of main results
		Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age			Interventions/comparisons/outcomes
Sciberras et al. (2015)	Cross sectional	ADHD:189	85%/15%	149 children with ADHD were on medication.	5–13 years	Working Memory Test Battery for Children (WMTB-C), CSHQ, Sleep self-report (SSR), Diagnostic Interview Schedule for children-IV, Socioeconomic Indexes for Area (SEIFA). Limitations: Reliance on subjective report measures, rather than objective, assessment of broad range sleep problems over past week, rather than specific difficulties on night before assessment, severe sleep problems in sample may have limited study's ability to detect relationships between sleep and working memory, majority children with ADHD on medication, might have influenced results.	To examine the relationship between sleep problems and working memory in children with ADHD.	As stated in the findings for each standard deviation increase in parent-reported bedtime resistance problems, working memory scores decreased by 3.4 points (95% CI: −6.3, −.6; $p = .02$), also for each standard deviation increase in child-reported sleep problems, working memory scores decreased by −4.2 points (95% CI: −7.0, −1.4; $p = .004$).
Cremone et al. (2018)	Case control	ADHD:18, Control:15	72%/28%	2 children with ADHD were on medication (abstained from medication for 48 hours prior to testing)	4–8 years	Diagnostic Interview Schedule for children-IV, Dot Probe task. Limitations: No sleep measure used in this study, although the effect of before and after sleep on the dot probe task was studied, no social-emotional tool used to measure individual differences that might affect reactions ADHD vs. the control children, small sample.	To determine whether emotional attention biases are evident in young children with clinically significant ADHD symptoms.	ADHD children had significant attention biases toward positive stimuli (happy faces shown in the dot probe task) ($p = .027$) whereas typically developing children had no significant attention bias for positive stimuli, ($p = .130$). Also positive attention bias was significantly greater in ADHD children as compared to typically developing children ($p = .008$).
*Saito et al. (2019)	Case control	ADHD:21(ADHD+ASD-10), Control:18	100%/0% (ADHD group) 50%/50% (Typically developing group) 75%/25% (ADHD+ASD).	ADHD children were ADHD medication naïve, however before the testing sleep sessions all ADHD patients and 2 typically developing children were administered pentobarbital (1.25–2.5 mg/kg wt or triclofos sodium (50–70 mg/kg wt).	7–12 years	Raven's Coloured Progressive Matrices (RCPM), Das-Naglieri Cognitive Assessment System (DN-CAS) and Mogras test with the Continuous Performance Test (CPT), Electroencaphalography (EEG), Swanson, Nolan, and Pelham IV scale (SNAP-IV), Parent-interview ASD Rating Scale (PARS-TR). Limitations: No girls in sample, EEG frequency analysis based on one 1-hour recording, frequency analysis in stage 2 may be different from that of all-night recording, medication induced-sleep group included only few individuals.	To evaluate the power of slow sleep spindles during sleep stage 2 to clarify their relationship with executive function, especially with attention, in children with ADHD and controls.	The relative power at 12 Hz was significantly higher in the ADHD + ASD compared to the TDC group at the frontal regions, additionally, the relative power at 12 Hz was significantly higher in the ADHD + ASD compared to the ADHD group ($p < .01$) and there was a significant correlation between the ratio of 12-Hz spindles and reaction time variability on CPT ($r = .368$, $p = .0242$).

(continued)

Table 5. (continued)

Authors/ publication year	Study design	Participant characteristics				Primary measures, limitations and biases	Objective	Summary of main results
		Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age			Interventions/comparisons/outcomes
Surratt et al. (2011)	Cross sectional	Edonotonsillar Hypertrophy: 56	47%/53%	Not currently medicated.	6–12 years	Actigraph, PSG, Subscales from the WISC-III, Wide Range Assessment of Memory and Learning (WRAML), Conner's Continuous Performance Test (CPT), Connor's Parent Behavior Rating Scale long form. Limitations: PSG the night after cognitive testing does not describe child's sleep night before testing, used visual inspection of actigraph graph in conjunction with the sleep diary to determine Time in Bed since no published criteria for children with sleep apnea.	To determine whether frequent movements over six nights during Time in Bed detected with wrist actigraphy predicted impaired cognitive and behavioral performance.	Frequency of movement during sleep predicted impaired vigilance (Min $>$ 5Mvts/night- R squared = .23, p = .00027) and (∑Movements Bed/ night- R squared = .19, p = .001) while consolidation of movements associated with impaired verbal (R squared = .16, p = .002) and memory skills (R squared = .21, p = .0035) and Obstructive Apnea Hypopnea Index (OAHI) was associated with more consolidation of movements (R squared = .38, $p <$ .0001).
Knight & Dimitriou (2019)	Case control	ADHD: 17, Control:20	82%/18%	2 children with ADHD were on medication.	5–11 years	Raven's Coloured Progressive Matrices, British Ability Scale (BAS-III) from which the digits forward subtest was used to assess short-term verbal memory, Conner's Parent Report Scale (CPRS), Conner's Continuous Performance Test (CPT), CSHQ, Actigraphy. Limitations: Small sample size, smaller number of females, ADHD subtype, obtaining more nights' actigraphy data (weekend-weekday) from participants would strengthen measure, attentional task is limited in its application to real-world context.	To investigate the relationship among sleep, ADHD behaviors, and attention in school-age children with and without a diagnosis of ADHD.	Poor sleep quality affects developmental subgroups in different ways, for example for ADHD children, poor sleep worsens their predisposed attentional deficit (actigraphy measured time in bed and sleep latency showed trends of prediction for accuracy in CPT task R2 = .46, p = .62), while for TD children it mimics ADHD behaviors (more sleep problems predicted a higher ADHD rating, R squared = .40, p = .05).
Hansen et al. (2013)	Case control	ADHD:38, ADHD + Anxiety disorder:25, Anxiety Disorder: 39, Controls: 35	63%/37%	Medication naive.	7–13 years	KSADS-PL, Children's Global Assessment Scale (CGAS), CBCL (Child Behavior Checklist) + TRF (Teacher Report Form), Wechsler Abbreviated Scale of Intelligence (WASI), CSHQ, Attention Network Test. Limitations: CSHQ not diagnostic tool for sleep disorders, no objective measure of sleep, diagnostic assessment based on parent reports only, no input from teachers collected, small sample size did not allow enough statistical power to identify relations between variables.	To examine associations between sleep problems and attentional and behavioral functioning in children with anxiety disorders, ADHD, combined anxiety disorder and ADHD and controls.	Sleep problems (increased CSHQ scores) were associated with reduced efficiency of the alerting attention system (warning signals had less effect in reducing reaction times during the test task) for all children (Beta coefficient of −1.84, p = .012) and with increased internalizing problems in children with anxiety disorders (p = .034).

(continued)

Table 5. (continued)

Authors/ publication year	Study design	Participant characteristics				Primary measures, limitations and biases	Objective	Summary of main results
		Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age			Interventions/comparisons/outcomes
Gruber et al. (2011)	Case control	ADHD:11, Control: 32	63%/37%	4 children with ADHD on medication (asked to abstain for 48 hours before testing)	7–11 years	Petersen's puberty development scale, CBCL, Socio-economic Status questionnaire, Epworth Sleepiness Scale, Sleep Logs, Actigraph, Conner's Continuous performance test (CPT). Limitations: Small sample size, PSG only used post-CPT performance during 1 week, not able to be used as measure of sleep quality and duration, rapid withdrawal of medications before baseline could have affected results, brain circuits involved in the attention and arousal systems could be focused on.	To assess impact of 1 hour of nightly sleep restriction over 6 nights on neurobehavioral functioning of children with ADHD and healthy controls.	Although the CPT scores of children in control group, deteriorated during the sleep restriction week, all scores nonetheless remained below the clinical cut off range of 60, however, for ADHD children, the significant deterioration of ~6-15 points in scores for Omission Errors, RT, change in RT, and Variability (@ $p = .05$ level) during the week of sleep restriction resulted in reduction in performance from subclinical levels of inattention to scores higher than or equal to a clinical cut off range of 60 on two-thirds of CPT outcome measures.
Bestmann et al. (2019)	Case control	ADHD:17, Control: 16 (children), 23 (adults)	100% males in all groups	12 children with ADHD were taking MPH but abstained for 48 hours prior to testing sessions.	9–12 years	Culture Fair Intelligence Test (CFT-20-R), battery for the assessment of attention (KITAP), PSG. Limitations: Small sample size for both the patient and the control group.	To investigate associations between sigma power during sleep and cognitive performance in healthy and ADHD children.	Only healthy children displayed a positive correlation between sigma power and reaction times (highest $r = .733$, @ $p < .005$ over the F4 region) and a negative association between IQ and sigma power (highest $r = .511$ (not alpha corrected), @ $p < .05$ over the P3 region) indicates a disturbed function of sleep in cognitive functions in ADHD.
Um et al. (2016)	Cross sectional	ADHD:28	78%/22%	Medication naïve.	6–12 years	Wechsler Intelligence Scale for Children-III (WISC-III), Matching Familiar Figure Test for Korean Children (MFFT-KC), Conner's Global Index-Parent version (CGI-P), PSG. Limitations: Lack of cognitive measures and PSG data for control group, sample sizes were small, majority subjects male, no subtyping of ADHD patients, first night effect of PSG not controlled.	To investigate the relationship between sleep parameters and cognitive function in drug-naïve children with ADHD.	Slow wave sleep, stage 2 sleep, REM sleep, and limb movement index with arousals are predictors of cognitive function in ADHD patients. Slow Wave Sleep (Beta coefficient of .40, $p = .019$) and Limb Movement with Arousals (Beta coefficient of .37, $p = .032$) best predicted Verbal IQ scores, whereas Conners Global Index scores was predicted by percentage of REM sleep (Beta coefficient of –.55, $p = .002$).

(continued)

Table 5. (continued)

Authors/ publication year	Study design	Participant characteristics				Primary measures, limitations and biases	Objective	Summary of main results
		Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age			Interventions/comparisons/outcomes
Zambrano-Sánchez et al. (2013)	Case control	ADHD:156, Control:111	67.4%/32.6%	Medication naive.	7–12 years	Paediatric Sleep Questionnaire (PSQ), Working Memory subscale from Wechsler intelligence scale for children (WISC-IV) to screen Executive Dysfunction (ED). Limitations: Larger population needed to obtain stronger conclusions, long term prospective follow-up, no PSG done for confirming presence of sleep disorders, covariates such as psychological, socio- cultural factors needed to reinforce results.	To determine the contribution of different types of sleep disorders in executive deficits among children with ADHD and typically functioning children.	A significant correlation was observed between PLMD frequency and ADHD-C type frequency ($r = .78$, $p = .05$) and between RLS frequency and ADHD-C and ADHD-H type frequency ($r = .65$, $r = .65$ respectively, $p = .05$), between ADHD-C children's with PLMD and coding values (WISC-IV) ($r = .75$, $p = .05$), between OSAHS and block design scores (WISC-IV) ($r = .64$, $p = .05$), and between ISH and digit values (WISC-IV) ($r = .57$, $p = .05$), for children with ADHD-I, significant correlations between ISH and each of coding ($r = .81$, $p = .05$), block designs ($r = .69$, $p = .05$), and digits scores ($r = .71$, $p = .05$).
Schmid and Wolke (2014)	Longitudinal	Infants:1120	50.6%/49.4%	Not mentioned.	Birth − 8.5 years	Neurodevelopmental Assessment, Tester's Rating of Child Behavior (TRCB), Kaufman's Assessment Battery for Children (K-ABC), Mannheimer Parent Interview (MPI), sleep was assessed through parental report. Limitations: Data collected in 1985–86. (standard of care has changed), sample consisted children referred to special neonatal care units after birth, results might not be generalizable to all infants requiring normal postnatal care, regulatory problems not assessed via diaries, quality of parent–child interaction at school age not assessed or controlled for.	To investigate if persistent Regulatory Problems (RP) during the preschool are precursors of ADHD and cognitive deficits in early childhood years.	RP at three measurement points (i.e., at 5, 20, and 56 months of age) were a predictor of lower IQ (K-ABC) at 8.5 years (small effect size: .17), poorer TEAM rating attention score (small effect size: .10), and of more activity (TRCB) (small effect size: .11), after controlling for confounding variables. RP at all three measurement points significantly increased the odds of inattention (MPI) (Odds Ratio (OR) 2.44), hyperactivity (MPI) (OR 3.09), and of a DSM-IV diagnosis of ADHD (MPI) (OR 3.32).

(continued)

Table 5. (continued)

Authors/ publication year	Study design	Participant characteristics: Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age	Primary measures, limitations and biases	Objective	Summary of main results: Interventions/comparisons/outcomes
Saletin et al. (2017)	Case control	ADHD: 7, Control: 14	72%/28%	ADHD children withdrawn from psychostimulant medication for 2 weeks before and during the in-lab study.	10–12.9 years	Actigraph, PSG, Motor Sequence Task (MST). Limitations: Differences in sleep EEG reported might reflect not difference in ADHD but rather in learning-dependent changes in EEG, subjects recruited to be free of disordered sleep thus study cannot address sleep disorders in ADHD's impact overnight improvements in motor learning, slow wave activity difference not assessed, participants withdrawn from psychostimulants throughout protocol, limiting ecological validity of their sleep.	To examine if the motor learning ability in ADHD children may be sensitive to spindle-frequency EEG activity expressed during sleep.	The ADHD group expressed lower power in the slow ($p = .023$, $d = -1.15$) and fast ($p = .049$, $d = -.98$) spindle bands, with the greatest deficit observed for slow spindle activity. ADHD group had impaired MST precision in the evening as compared to the TD group (Wald-$\chi^2 = 3.90$, $p = .048$, $d = 1.33$), this effect was ameliorated in the morning following sleep (Wald-$\chi^2 = .02$, $p = .88$, $d = .34$) and MST precision was positively associated with slow spindle activity for the ADHD group ($\beta = 8.71$, $p = .003$), but not for TDCs ($\beta = .50$, $p = .82$).
Prehn-Kristensen et al. (2011)	Case control	ADHD: 16, Control: 16	100%/0%	12 children with ADHD took medication (MPH) however they discontinued its intake 48 hours prior to each testing session.	9–12 years	K-SADS-PL, CBCL, Culture Fair Intelligence Test Revised Vision (CFT-R), Diagnosticum für Cerebralschädigung (DCS), Pittsburgh Sleep Quality Index (PSQI), Procedural Memory Task, KITAP, PSG. Limitations: Limited sample size, no female participants, no social- emotional functioning variable is measured that might effect learning.	To investigate sleep associated consolidation of procedural memory in ADHD children (sleep and wake conditions).	Children with ADHD showed improvement in motor skills after sleep compared to the wake condition [sleep condition: 202.4 ± 18.0; wake condition: 163.1 ± 14.3; $t(15) = 2.46$, $p = .026$] and sleep-associated gain in reaction times was positively correlated with the amount of sleep stage 4 ($r = .719$, $p = .002$) and REM sleep-density ($r = .589$, $p = .016$) in ADHD.
Prehn-Kristensen et al. (2017)	Case control	ADHD: 16 (comorbid Oppositional Defiant Disorder), Control:16	100%/0%	11 children with ADHD took medication (MPH) however they discontinued its intake 48 hours prior to each testing session.	8–11 years	Sleep Self-report questionnaire, CSHQ, CBCL, Culture Fair Intelligence Test 20-Revised Version (CFT 20-R), Picture recognition task, eye-tracking and pupillometry, PSG. Limitations: Non-social stimuli could be used since faces can act as emotional stimuli, a balanced number of trials across immediately and delayed recognition measurement should be considered, inclusion of psychophysiological parameters during encoding are required.	To investigate the influence of sleep on the picture recognition of emotional faces and their affective regulation in children with ADHD and comorbid ODD (ADDH + ODD) in comparison to healthy children (sleep and wake conditions).	Pupillometry and behavioral data revealed that healthy children benefited from sleep compared to wake with respect to face picture recognition ($p < .001$) ; in contrast recognition performance in patients with ADHD + ODD was not improved after sleep compared to wake ($p < .001$).

(continued)

Table 5. (continued)

Authors/ publication year	Study design	Participant characteristics				Primary measures, limitations and biases	Objective	Summary of main results
		Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age			Interventions/comparisons/outcomes
Weisner et al. (2017)	Case control	ADHD:17 (comorbid Conduct Disorder & Oppositional Defiant Disorder), Control:17	100%/0%	13 children with ADHD took MPH medication, but discontinued 48 hours prior to testing sessions.	8–12 years	Culture Fair Intelligence Test Revised Version (CFT-20-R), PSG, CSHQ, Sleep logs, KITAP (measuring attention), Sleep Self-report questionnaire, Pubertal Development Scale (PDS), Probabilistic Learning and Reversal task, Self-Assessment Manikin (SAM). Limitations: small sample, participants with comorbid social behavior disorder pose diagnostic entity separate from patients with only ADHD, results could be affected by circadian influences on learning performance, alertness, mood (valence, arousal).	To study if sleep fosters consolidation of behavior learned by probabilistic reward and whether ADHD patients with comorbid disorder of social behavior show deficits in this memory domain.	The drop in performance (in the probabilistic reward task) between sleep and wake in the ADHD children ($p = .264$, $d = .28$) did not differ but this difference was stronger during sleep than during wake in the control participants ($p = .042$, $d = -.51$), and this double difference was significant ($p = .028$, $d = .79$), which points that only the control participants showed sleep-dependent consolidation of rewarded behavior. For ADHD children there were only non-significant correlations of the performance drop with REM sleep ($r = .322$, $n = 17$, $p = .207$, $d = .68$) and non-REM sleep ($r = .138$, $n = 17$, $p = .597$, $d = .28$), in contrast, control participants showed a significant ($r = .441$, $n = 17$, $p = .076$, $d = .98$) correlation of performance drop with non-REM sleep (not with REM sleep), this points that retention of rewarded behavior over sleep did not correlate with sleep, especially not with REM sleep for the ADHD children, which was not the case for the control children.
Yürümez and Kılıç (2016)	Case control	ADHD:46, Control:31	100%/0%	Medication naïve.	7–13 years	CSHQ, Conner's Parent Rating Scale– Revised Long Form, Paediatric Quality of Life Inventory (PedsQL), Conner's Teacher Rating Scale–Revised Long Form, WISC-R(Wechsler Intelligence Scale for Children-Revised), K-SADS-PL. Limitations: small sample size, lack of PSG data from all participants to rule out primary sleep disorders, parents' sleep habits and family stress not assessed, parents' ratings of sleep problems for children may be subject to rater bias and inaccuracy.	To assess the sleep behaviors, sleep problems and its frequency, and to evaluate the effect of sleep problems on quality of life for ADHD and control children.	PedsQL -Psychosocial subscale scores, completed by parents, are found significantly different for ADHD and control group (Mean 62, SD- 15.6 vs. Mean 79, SD- 10.6, $p < .001$) and between those children having sleep problems and those without sleep problems (Mean 62, SD- 15.6 vs. Mean 79, SD- 10.6, $p = .033$).

(continued)

Table 5. (continued)

Authors/ publication year	Study design	Participant characteristics				Primary measures, limitations and biases	Objective	Summary of main results
		Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age			Interventions/comparisons/outcomes
San Mauro Martín et al. (2018)	Case control	ADHD: 41, Control: 48	68.3%/31.7%	Not mentioned.	6–12 years	The Mediterranean Diet Quality Test for Children and Adolescents (KIDMED Index), Anthropometry, Physical Activity, Sedentary behaviors, Short Form Health Survey (SF-36), Sleep quality assessed by recording sleep duration (weekends/ weekdays). Limitations: Findings were not completely in line with previous literature in the topic and this could be due to the limited sample size.	To determine the association between environmental, nutritional, and body composition factors that may affect the pathogenesis and symptomatology of ADHD patients.	Low adherence to a Mediterranean diet might play a role in ADHD development. The average hours of sleep per week differed between ADHD and control group ($p = .031$), so did sedentary behaviors (time spent watching television and/or using computer/mobile/tablet ($p = .238$) and reading ($p < .001$)), same was the case for KidMed's final score ($p = .004$) and lastly for food items, observed in fish ($p = .001$), cereal ($p = .002$), no breakfast ($p = .007$), and commercially baked goods ($p = .01$) consumption.
Tong et al. (2016)	Cross sectional	Total children: 785 (ADHD symptoms 82)	52%/48%	Not mentioned.	9–13 years	Physical Activity Questionnaire for Older Children (PAQ-C), sleep environment, bedtime activities and screen time records, Child Eating Behavior Questionnaire (CEBQ), ADHD-RS-IV. Limitations: Study used questionnaire not interview-based assessments to assess ADHD, physical activity measured by self-reported questionnaire rather than accelerometers, it is a cross sectional study, limiting ability to indicate causality.	To study the associations between ADHD symptoms in children and their associated lifestyle.	Children with ADHD symptoms showed more eating behaviors ($\beta = .04$, $p < .01$), bedtime activities ($\beta = .05$, $p < .05$) and more electronic devices in bedroom ($\beta = .01$, $p < .01$) after controlling for children's gender, parents and children's age, parents' education level and annual household income.
Tong et al. (2018)	Cross sectional	Children: 934 (ADHD symptoms 82)	53%/47% (total participants), Within ADHD group: 14%/6%	Not mentioned.	9–12 years	ADHD Rating Scale-IV, Children's Sleep Habits Questionnaire, CBCL, Diet behaviors, Screen time. Limitations: Cross-sectional design, limiting study's ability to draw causal inferences, sleep hours and sleep problems were assessed by parent-reported rating scales rather than objective measures, study used a questionnaire and not an interview-based assessment to assess ADHD, data on medication not collected.	To determine the moderating effects of bedtime activities and depression/anxiety symptoms on the relationship between ADHD symptoms and sleep problems.	Bedtime activities and emotional problems had important moderating effects on the relationship between ADHD symptoms and sleep problems (for example interaction between screen time and ADHD predicted sleep duration on weekends ($\beta = -.111$, $p < .05$), sleep onset delay ($\beta = -.058$, $p < .05$) R squared = .094, and sleep-disordered breathing ($\beta = -.052$, $p = .057$) R squared = .074 and eating before bedtime played a moderating role in the relationship between ADHD and night waking ($\beta = .061$, $p < .1$).

(continued)

Table 5. (continued)

Authors/ publication year	Study design	Participant characteristics				Primary measures, limitations and biases	Objective	Summary of main results
		Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age			Interventions/comparisons/outcomes
Peralta (2018)	Longitudinal	Children: 817	51%/49%	Not mentioned.	4–7 years	Conners' Parent Rating Scales and the Strengths and Difficulties Questionnaire, parental report of time spent watching TV, sleeping and engaging in cognitive stimulating activities. Limitations: Use of parental questionnaires to assess variables and thereby misclassification subject to inaccuracy, children who had longer breastfeeding, higher maternal IQ, and higher parental education levels than those who did not participate, no information on pharmacological or nonpharmacological.	To analyse associations between time spent sleeping, watching TV, engaging in cognitively-stimulating activities, and engaging in physical activity (assessed at 4 years) and ADHD symptoms and behavior problems, both assessed at 7 years, in ADHD-free children at baseline.	A shorter sleep duration and less time spent in cognitively stimulating activities were associated with an increased risk of developing ADHD symptoms ($p = .006$), in addition, engaging in less cognitively stimulating activities at 4 years was associated with an increased risk of developing ADHD symptoms ($p < .001$) and developing behavioral problems ($p = .005$).
Jackson and Vaughn (2017)	Longitudinal	Children: 3800	52%/48%	Not mentioned.	1–10 years	Parental reports for sleep duration, sleep problems, preteen delinquency, Conner's Teacher's Rating Scale-Revised Short Form, CBCL. Limitations: Limited items pertaining to sleep quality, measures (with the exception of teacher plus parent report of ADHD), were based on parental/ caregiver report, continuation of delinquency beyond preteen years could not be definitively determined due to data limitations, lack of experimental design, high-risk families overrepresented in sample.	To examine associations between sleep patterns, ADHD, and delinquency.	There is a significant association associations between ADHD symptomatology, ADHD diagnosis, and preteen delinquency, with odds ratios ranging from 2.38 to 8.10 ($p < .001$), additionally accounting for both ADHD symptomatology and ADHD diagnosis, the negative association between sleep duration and preteen delinquency is significant (OR = .85, $p = .011$).

contributions to parental reports of bedtime resistance, whereas an evening circadian tendency contributed to parental reports of sleep onset delay and to PSG-measured sleep-onset latency (Gruber, Michaelsen, et al., 2012).

When considering comorbidities, it was found that children with ADHD ($n=392$) and externalizing comorbidities had higher reported night waking than children with ADHD alone (Lycett, Mensah, et al., 2015). Internalizing and externalizing comorbidities such as separation anxiety, social phobia, specific phobia, obsessive/compulsive disorder, dysthymia or conduct disorder in children with ADHD were found to be associated with moderate/severe sleep problems (Lycett, Sciberras, et al., 2015; Thomas et al., 2018, ADHD $n=299$). A longitudinal investigation over an average of 3.8 years found associations between sleep patterns (shorter sleep duration, later sleep timing preference, poorer sleep continuity, and worsening daytime sleepiness) and longitudinal changes in five psychiatric symptoms severity measures (mania, depression, anxiety, mood lability, and inattention/ externalizing) in 267 high risk youth aged 10 to 18 years (including 145 ADHD individuals) (Soehner et al., 2019).

Consequences for cognitive and executive functioning. Moreau et al. (2013) reported a strong relationship between reduced total sleep time (measured through actigraphy) and increased executive functioning concerns among 6 to 13-year-old children with ADHD ($n=43$) in a parent-reported scale measuring emotional/behavioral and cognitive regulation. In a longitudinal study ($n=271$) it was found that children for whom more sleep problems were reported at 3 years, with executive control deficits (working memory and inhibitory control) found at 4.5 years, had greater ADHD symptomatology in 4th grade, and children with both sleep problems and executive control deficits experienced more hyperactivity than those with high executive control functions (Kidwell et al., 2017). Further, it was reported that the reduced inhibitory control found among 18 children with ADHD (in an inhibition-attention task), remained unchanged after sleep, whereas inhibitory control improved significantly among typically developing children ($n=15$) after sleep (Cremone et al., 2017). REM sleep theta activity was positively associated with morning inhibitory control in TD children but not in children with ADHD, even with REM theta activity being significantly greater in the ADHD group compared to the TD group (Cremone et al., 2017). However, Sciberras et al. (2015) reported most parent-reported sleep problem domains were not associated with the ADHD child's working memory performance (measured through objective working memory test), with the exception of bedtime resistance and night-waking difficulties ($n=189$, 5–13 years). Children with ADHD symptoms ($n=18$) exhibit attention biases toward positive, but not negative, stimuli whereas TD children ($n=15$) do not direct attention toward or away from positive or negative stimuli and these emotional attention biases were unchanged following overnight sleep in children with ADHD symptoms (Cremone, Lugo-Candelas, et al., 2018).

Analysis of EEG frequency during stage 2 sleep, revealed high voltage spindles in most children with ADHD ($n=21$, 7–12 years), dominant at the frontal pole and frontal regions along with slow spindle activity in children with ADHD ($n=11$), greater than in typically functioning children ($n=18$) and this frontal slow-spindle activity had a positive correlation with the variability of reaction times in the Continuous Performance Test (CPT) results measuring attention (Saito et al., 2019). Further, a significant relationship between lower actigraphy-measured sleep efficiency and poorer attentional performance reflected through increased variability in reaction times on a CPT was found among 43 children with ADHD aged 6 to 13 years (Moreau et al., 2013). In another study ($n=56$, not on children diagnosed with ADHD), CPT performance indicated that high reaction time scores were associated with more movements during sleep (measured through night actigraphy; Surratt et al., 2011). On similar lines, it was found that poor sleep was predictive of increased ADHD trait behaviors in a TD sample ($n=20$, 5–11 years), but were not associated with impaired attentional capacity measured through the CPT (Knight & Dimitriou, 2019). However, in the same study poor sleep (parasomnias, time in bed, sleep latency as revealed through CSHQ and actigraphy) is predictive of reduced attentional capacity in the ADHD sample ($n=18$, 5–11 years) with no predictive power toward ADHD-related behaviors, demonstrating that problems with sleep may exert impact on typically developing and clinical childhood populations in distinct ways (Knight & Dimitriou, 2019).

For a sample with ADHD, TD and ADHD + Anxiety groups, it was found that for the whole sample ($n=102$, aged 7–13 years), increases in total reported sleep problems predicted lower alerting scores on an Attention Network Test (a computer based test assessing the capacity to achieve and maintain a state of high sensitivity to incoming stimuli; Hansen et al., 2013). For sleep duration, it was found that for 7 to 11-year-old children with ADHD ($n=11$), sleep shortening of 1 hour for 6 nights was associated with deterioration in CPT performance from subclinical to clinical ranges of scores, and that this deterioration in TD children ($n=32$) did not reach clinical range; this may indicate that sleep shortening exerts a significant negative impact on neurobehavioral functioning in children with ADHD (Gruber et al., 2011).

As an indicator of disturbed sleep's effect on cognitive performance, an association between stage 2 sleep oscillations (characterized by sigma power) and cognitive performance was found in children with ADHD ($n=17$, aged 9–12 years), when a negative correlation between sigma power in the parietal area and IQ was found (Bestmann et al., 2019). Similarly, Um et al. (2016) found that the percentage of stage 2 sleep in ADHD ($n=28$, 6–12 years) was

negatively correlated with verbal IQ (WISC-III) and predicted response latency time in an evaluation of cognitive reflection-impulsivity. In another study ($n=56$, 6–12 years), actigraphy-measured bed time movements predicted poorer performance on the cognitive assessment subscales of vocabulary and similarities (WISC-III subtests for assessing retrieval of learned verbal information and abstract reasoning/concept formation respectively) and general memory index score (assessing immediate and short term verbal memory) on a standardized memory and learning test (Suratt et al., 2011). Zambrano-sánchez et al. (2013) found correlation between the presence of specific sleep disorders and WISC-IV measurements among 7 to 12 year old children with individual subtypes of ADHD ($n=156$); children with ADHD-combined presentation showed significant correlations between periodic limb movements during sleep and coding subtest value (measuring processing speed), between obstructive sleep apnea-hypopnea syndrome and block design values (measuring Perceptual reasoning) and between inadequate sleep hygiene and digit span subtest values (measuring working memory), whereas children with ADHD-Hyperactivity showed significant correlations between sleep apnea and digit space score and inadequate sleep hygiene and block design scores; and finally, children with ADHD-Inattentive showed significant correlations between inadequate sleep hygiene and each of coding, block designs, and digits span scores (Zambrano Sanchez et al., 2013). A prospective study (birth to 8.5 years, $n=1120$) found persistent regulatory problems in sleep behaviors during infancy predicted lower IQ, increased attention deficits as observed during the test situation, and considerably increased odds of a ADHD diagnosis during preschool years (Schmid & Wolke, 2014).

When assessing procedural learning through motor sequence task (MST) in childhood ADHD (10–12 years), an association was found between slow spindle sleep EEG frequency activity and overnight improvement in MST precision; specifically, MST precision was positively associated with slow spindle activity for the ADHD group ($n=7$) but not for TD group ($n=14$) (Saletin et al., 2017). Similarly, another study found that sleep-associated improvements in reaction times in a motor skills task were positively correlated with the amount of sleep stage 4 and REM-density in children with ADHD (n-16) but not in the control group (n-16), suggesting that sleep in ADHD normalizes deficits in daytime procedural memory (Prehn-Kristensen et al., 2011). Prehn-Kristensen et al. (2017) found that in a task of rating faces for emotional content, pupilometry and behavioral data revealed among 8 to 11-year-old children with ADHD and comorbid ODD ($n=16$) performance did not improve after sleep, unlike the post-sleep improvement noted in the matched controls ($n=16$). For consolidation of behavior learned by probabilistic reward, it was found that 8 to 12-year-old children with ADHD ($n=7$) do not show sleep-dependent consolidation of rewarded behavior, whereas typically functioning children ($n=17$) consolidate rewarded behavior better during a night of sleep, and the level of consolidation correlates with non-REM sleep (Weisner et al., 2017).

Lifestyle factors and sleep in ADHD. Yürümez and Kılıç (2016) reported that pediatric quality of life scores in 7 to 13-year-old children with ADHD (n-=46) with sleep problems were significantly lower than control group children ($n=31$), with ADHD children receiving worse parent ratings in the scale's Psychosocial subscale. The relationship between sleep-related problems and dietary intake in children with ADHD ($n=88$, 6–13 years) has been examined, with higher consumption of fats (mono- and polyunsaturated), energy, carbohydrates and sugar being positively associated with sleep disturbances (Blunden et al., 2011). Moreover, ADHD has been associated with lower adherence to a diet of fish, pulses, pasta or rice and a significant difference between ADHD ($n=41$, 6–12 years) and controls group ($n=48$) for sedentary behaviors and sleep quality (by assessing hours of sleep on weekdays and weekends) (San Mauro Martín et al., 2018). Further, an increased risk of obesity in children with ADHD symptoms ($n=82$, 9–13 years) was found to be associated with overuse of electronic devices, eating while using electronic devices, and delaying bedtimes to snack and use electronic devices (Tong et al., 2016). Considering screen time, children with more ADHD symptoms ($n=82$, 9–12 years) and screen time before bedtime tended to have shorter sleep times on weekends (Tong et al., 2018). A longitudinal study ($n=817$) where parent rated sleep, TV watching, physical activity and engaging in cognitively stimulating activities at 4 years and ADHD screener and behavioral questionnaire at 7 years was investigated among a cohort, found that shorter sleep duration and less time spent in cognitively stimulating activities were associated with increased risk of developing ADHD symptoms and behavioral problems (Peralta et al., 2018).

Sleep-Related Interventions and their Impact on Sleep and Daytime Function in ADHD

Details of the studies reviewed on sleep interventions and their impacts on daytime function in ADHD are detailed in Table 6. When the behavioral and neurocognitive effects of neurofeedback were compared to stimulant medication and physical activity in 7 to 13-year-old children with ADHD ($n=92$) at 6 months' follow-up parents and teacher reported improvements for neurofeedback on sleep quality (assessed through the Sleep Disturbance Scale; Geladé et al., 2018). A study assessing the effectiveness of cognitive rehabilitation of response inhibition in improving the quality of sleep and behavioral symptom of children with ADHD (7–12 years)

Table 6. Summary of Thirteen Included Studies Focusing on Non-Pharmacological Interventions and Their Impact on Sleep and Overall Functioning in Children with ADHD.

		Participant characteristics						Summary of main results
Authors/ publication year	Study design	Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age	Primary measures, limitations and biases	Objective	Interventions/comparisons/ outcomes
Geladé et al. (2018)	Randomized control trial	ADHD:92	76%/24%	36 children with ADHD were allocated to medication intervention (MPH), however at 6-month follow-up, 21 were still using medication and 6 had discontinued.	7–13years	Auditory oddball, stop-signal, and visual spatial working memory tasks, Strength and Difficulty Questionnaire (SDQ) and Strengths and Weaknesses of ADHD symptoms and Normal Behavior Scale (SWAN), Sleep Disturbance Scale for children (SDSC). Limitations: Naturalistic controlled follow up, so potentially unknown factors might have improved outcomes, small sample limiting statistical power, statistical power of sensitivity analyses was reduced due to smaller sample size.	To compare behavioral and neurocognitive outcomes at a 6-month naturalistic follow-up of a randomized controlled trial for neurofeedback (NFB), methylphenidate (MPH), and physical activity (PA).	Improved inhibition in MPH compared to NFB ($p < .001$ after intervention and $p = .040$ at follow-up) and faster response speed in NFB compared to PA during the stop-signal task ($p = .012$). Results demonstrated comparable improvements in sleep quality for all interventions.
Yazdanbakhsh et al. (2018)	Quasi-experimental intervention study	ADHD: 20 (10 in treatment group, 10 in control group)	60%/40%	Currently not medicated.	7–12years	Conner's Parents and Teacher's Rating Scale (CPRS), Pittsburgh Sleep Quality Index (PSQI). Limitations: Limited sample size, absence of control group with another treatment form and also follow up outcomes for interventions.	To measure the effectiveness of 12 sessions of cognitive rehabilitation of response inhibition in improving the quality of sleep and behavioral symptom of children with ADHD.	Intervention bettered the quality of sleep (effect size: .63, $p < .001$) and behavioral symptoms in ADHD (effect size: .58, $p < .001$).
Hvolby and Bilenberg (2011)	Case-control intervention study	ADHD: 21 control: 21	90%/10%	Children with ADHD were on medication (Stimulant, Atomoxetine, Dexamphetamine, Stmulant + Atomoxetine, Melatonin, Alternative medicine).	8–13years	Actigraphy recordings, parent completed sleep diary. Limitations: Relatively small sample size, short lenghth of time for use of blanket, subtypes of ADHD not identified in sample, effect of medication not examined.	To assess the effect of using a ball blanket for 14 nights and 14 nights without it (pre & post), through actigraphy recordings and parental reports.	Results indicated improvements among ADHD children in sleep onset latency (the average sleep onset latency was 23.1minutes, which fell to 14.0minutes when using the Ball Blanket—a fall of 39.4%, $p < .002$), the proportion of single nights when more than 30minutes were spent falling asleep fell from 27.7% to 14.8% when using the blanket($p < .003$), which is the same level as the healthy control children, parents reported that with the use of the ball blanket sleep onset latency fell from an average of 36.7 to 26.9minutes, which is an improvement of 26.7% ($p < .001$).

(continued)

Table 6. (continued)

Authors/ publication year	Study design	Participant characteristics				Primary measures, limitations and biases	Objective	Summary of main results
		Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age			Interventions/comparisons/ outcomes
Hiscock et al. (2015)	Randomized controlled trial	ADHD: 244	84%/16%	211 children were on medication (MPH, Atomoxetine and Clonidine).	5–12years	ADHD rating scale, CSHQ, Actigraphy, Strengths and Difficulties questionnaire, Paediatric quality of life inventory 4.0), Daily Parent Rating of Evening and Morning Behavior (DPREMB),working memory test battery, parent mental health (depression anxiety stress scales). Limitations: Parents not blinded about randomization results, leading to possible overestimation of improvements, response rate at 3 months was low.	To study the effect of a sleep hygiene Intervention and standardized behavioral strategies in 2 fortnightly sessions and a follow up phone call on the ADHD child's sleep problems, ADHD symptoms, functionality and working memory.	A brief behavioral sleep intervention modestly improved the severity of ADHD symptoms ((at 3 and 6 months follow-up) $p = .03$, effect size −.3, and $p = .004$, effect size −.4, respectively), compared to children in the control group, intervention children had fewer moderate-severe sleep problems at 3 months (56% vs. 30%; adjusted odds ratio .30, $p < .001$) and 6 months (46% vs. 34%; adjusted odds ratio- .58; $p = .07$), Working memory was found to be higher in the intervention children compared with control children at 6 months. And daily sleep duration was higher in the intervention children at 3 months (effect size .2) and 6 months (effect size .3).
Papadopoulos et al. (2019)	Randomized control trial	ADHD comorbid ASD: 61 (28 in treatment group and 33 in control group)	85%/15% (Intervention) 90%/ 10 % (usual care)	53 children with ADHD were on medication (MPH (long and short acting), Atomoxetine and Clonidine).	5–13years	CSHQ, Strength and Difficulties questionnaire, ADHD rating scale- IV, Paediatric Quality of Life questionnaire, Depression Anxiety Stress scale (for parents). Limitations: Reliance on parent's reports for ASD assessment, rather than objective assessment tool, sample had average intellectual ability, so children with clinical level deficit not explored, parents not blinded for interventions possibly affected ratings, small sample size.	To study the efficacy of a brief behavioral sleep intervention.	Results showed improvements in sleep problems (CSHQ total score, 3 months (effect size- .7) other sub scales also showed improvements such as sleep onset delay, effect size- .9 at 3 months post intervention; sleep duration, effect size- .8 at 6 months post intervention and parasomnias, effect sizes- .6 and .7 at 3 and 6 months post intervention).

(continued)

Table 6. (continued)

Authors/ publication year	Study design	Participant characteristics: Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age	Primary measures, limitations and biases	Objective	Summary of main results: Interventions/comparisons/ outcomes
Bériault et al. (2018)	Exploratory Intervention study	ADHD:20, ADHD+Anxiety:20, Anxiety: 20, Control: 9	73%/27%	17 children with ADHD were on medication, 18 children with ADHD+ Anxiety were on medication (Psychostimulants, Atomoxetine and Risperdone).	8–12years	CSHQ, Anxiety Disorders Interview Schedule for children, ADHD rating scales- IV, Multidimensional Anxiety Scale for children (MASC). Limitations: Limited sample size, groups not compared on socio-economic variables, sleep was assessed by parent questionnaires, rather than objective measures.	To examine the effect of cognitive-behavioral therapy (CBT) for anxiety on sleep problems in ADHD children with comorbid anxiety disorders and to examine the sleep problems in children with ADHD and ADHD+Anxiety.	Total score and number of problems in the CSHQ had significantly higher values in each clinical group compared with the control group ($p < .001$ & $p < .05$ respectively), anxiety related to sleep were significantly greater in the three clinical groups compared with the controls ($p < .01$) and CBT led to significant outcomes for daytime somnolence (effect size: .36), Total score in CSHQ (effect size: .36) reduction in the first order severity score in the Anxiety Disorders Interview Schedule (effect size: .93), Total score for MASC (effect size: .60).
Keshavarzi et al. (2014)	Randomized control trial	ADHD: 40 (20 in control and 20 in trial)	95%/5%	All children with ADHD were on medication (MPH).	8–13years	CSHQ, KID-Screen, parent view and children view, Strength and difficulties questionnaire. Limitations: Limited sample size, no cognitive or ADHD-related assessments, no objective sleep assessment, parents not blinded for intervention group.	To study the effect of 12week randomized control trial for sleep training on emotional, social and behavioral functioning in children withADHD.	Intervention group improved sleep quantitatively and qualitatively ($p < 05$), intervention receiving children reported improvements in mood, emotions, and relationships ($p < .05$). Parents reported that their children improved in physical and psychological wellbeing, mood, emotions, relationships, and social acceptance ($p < .05$).
Peppers et al. (2016)	Intervention study	ADHD: 53 (23: treatment group)	57%/43%	All children with ADHD were on medication (MPH, Amphetamine and non-stimulant).	5–11years	CSHQ, Vanderbilt ADHD parent scale. Limitations: Small sample for intervention from the full number of participants.	To study the effect of a 20-week pilot project using a sleep hygiene routine to assess sleep and ADHD symptoms.	The CHSQ and Vanderbilt scores indicated a significant improvement in sleep quality and reduction in ADHD symptoms after implementation of the sleep hygiene routine (CHSQ: $p < .001$, $d = .928$; Vanderbilt Questions 1–9: $p < .001$, $d = .473$; Vanderbilt Questions 10–18: $p = .004$; $d = .329$).
Corkum et al. (2016)	Randomized control trial	ADHD: 61 (30 in treatment group and 31 in waitlist control group)	46%/54%	14 children with ADHD were on medication.	5–12years	Actigraphy, CSHQ, CBCL, Sleep Evaluation Questionnaire, K-SADS-PL. Limitations: Parents were not blind to the intervention conditions which may have affected their ratings, adherence to actigraphy was low, limiting power of analysis, small sample size.	To study the effect of five-session manual and weekly telephone coach support on the children's sleep and psychosocial functioning.	Results indicated improved sleep onset (effect size: .23, $p = .017$) improved parent rated CBCL internalizing (effect size: .30, $p < .001$) and externalizing scores (effect size: .27, $p < .001$).

(continued)

Table 6. (continued)

Authors/ publication year	Study design	Participant characteristics				Primary measures, limitations and biases	Objective	Summary of main results
		Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age			Interventions/comparisons/ outcomes
Pelsser et al. (2010)	Randomized control trial	ADHD: 27 (15 in treatment group and 12 in control group)	76%/34% (Intervention group), 81%/19%	Medication naive.	3.8–8.5 years	Pre-post assessment via Physical Complaints Questionnaire (includes 2 sleep questions). Limitations: Older children would better describe physical symptoms, sleep related movements disorders not explored in the sample, ADHD subtype not explored.	To investigate the effects of a 5 week elimination diet on physical and sleep complaints in children with ADHD.	The number of physical (headaches or bellyaches, unusual thirst or unusual perspiration) and sleep complaints was significantly decreased in the diet group compared to the control group ($p < .001$), with a reduction in the diet group of 77% ($p < .001$, effect size = 2.0) and in the control group of 17% ($p = .08$, effect size = .2).
Galán et al. (2017)	Intervention study	ADHD: 6, ASD: 7, Cerebral Palsy: 9	50%/50% (ADHD group), 42%/58% (ASD group), 55%/45% (Cerebral Palsy group)	Children with ASD: Risperidone, Aripiprazole and Paliperidone, Cerebral palsy took low doses of Benzodiazepine, Tryhexyphenidyl, Polyethylene or Antihistamines and patients with ADHD took Methylphenidate, long-acting Methylphenidate or Fluoxetine.	ADHD group: mean-8.67(SD-2.73) years	Actigraphy records. Limitations: Small sample size, not a randomized controlled trial.	To study the effect of Tryptophan enriched antioxidant cereals on sleep problems among children with ADHD,ASD and Cerebral Palsy.	Assumed sleep was significantly higher when the target diet was taken for dinner rather than only breakfast, or control, or basal week ($p < .05$), sleep latency levels were significantly ($p < .05$) lower than control levels when children took target diet for dinner, Sleep efficiency levels were significantly ($p < .05$) higher in diet taken in Dinner-week than in basal, control and diet taken in breakfast-week and immobile time improved ($p < .05$) in children with ADHD ingested tryptophan-enriched cereals at dinner than in control week.
Yehuda et al. (2011)	Intervention study	ADHD: 78(40 in treatment group and 38 in placebo group) Control: 22	100%/0%	Not	9–12 years	Questionnaire completed and hemoglobin level tested pre-post treatment. Limitations: No questionnaire or tool to assess other psychological variable.	To the study the effect of a 10 week polyunsaturated acid mixture on cooperation, mood, concentration, homework preparation, fatigue and sleep quality among ADHD children.	Results indicated significant improvement in quality of life, ability to concentrate, sleep quality and hemoglobin levels ($p < .05$ & $p < .01$ level of significance).
Sonne et al. (2016)	Intervention study	ADHD: 13	70%/30%	7 children with ADHD were on medication	6–12 years	The Daily Assessment Application (DAA), ADHD Rating Scale-IV, CSHQ. Limitations: Small sample.	2-week intervention with smartphone based application for bedtime routines/2 week baseline phase.	Use of MOBERO was associated with a 16.5% reduction in core ADHD symptoms $p < .05$, Cohen's d = .73 and an 8.3% improvement in the child's sleep habits ($p < .05$, Cohen's d = .67).

revealed significantly lower scores on the Pittsburgh sleep quality index (PSQI) and on the three features of hyperactivity, impulsivity and attention deficit (measured through parent rated Conner's questionnaire) for the treatment group ($n=10$) compared to the control group ($n=10$) (Yazdanbakhsh et al., 2018). With regard to techniques based on sensory integration, Hvolby and Bilenberg (2011) studied the effect of using a Ball Blanket aimed at stimulating both the sensation of touch and the sense of muscle and joint (and thereby leading to sensory impressions that transmit inhibitory impulses to the central nervous system) on sleep of children with ADHD ($n=21$, 8–13 years). The findings revealed that the use of Ball Blankets for 14 days improves sleep onset latency, individual day-to-day variation and number of awakenings to a level comparable with those found in the healthy control children ($n=21$) and reduces the frequency of nights in which the child spends more than 30 minutes falling asleep from 19% to 0%.

The impact of a behavioral sleep intervention in children with ADHD ($n=244$, 5–12 years) in a randomized controlled trial revealed significant parent-rated decrease in moderate/severe sleep problems at 3 months' post treatment; daily sleep duration measured by actigraphy was also increased in the intervention group children, and approximately a half to one-third of the beneficial effects of the intervention on ADHD symptoms was mediated through improved sleep at 3 and 6 months follow-up (Hiscock et al., 2015). A brief behavioral sleep intervention, comprising of two clinical consultations and a follow-up phone call covering sleep hygiene and standardized behavioral strategies, in 5 to 13 years old children with ADHD with comorbid Autism Spectrum Disorder, led to large improvements in parent-rated sleep problems at 3 and 6 months for the treatment (n-28) group compared with the usual care ($n=33$) group (Papadopoulos et al., 2019). Further, the intervention group ($n=28$) also had small to moderate improvements in parent-rated psychosocial quality of life, ADHD symptom severity and child behavior (Papadopoulos et al., 2019). When 20 children (8–12 years) in each group for ADHD, Anxiety and ADHD + Anxiety were treated with cognitive behavioral therapy for anxiety, this lead to significant decrease in anxiety along with improvement in sleep latency and marginal decrease in total amount of sleep problems for the treatment groups (Beriault et al., 2018).

Keshavarzi et al. (2014) showed that a 12-week sleep training program followed by parents of children with ADHD (8–13 years) led to significant improvements over time for the intervention group ($n=28$) in sleep problem areas such as being afraid of sleeping in the dark, sleeping alone, sleeping too little, sleeping the right and the same amount, bed wetting and sleeping restlessness, along with decrease in the duration of awakenings after sleep onset and increases in total sleep time. Parents of children in the intervention group also reported improved in physical and psychological well-being, moods and emotions, parent relations and home life, school environment, and social acceptance for their children. Peppers et al. (2016) investigated the effect of a 20-week intervention involving following a patient specific sleep hygiene routine to promote sleep and reduce ADHD symptoms among children (5–11 years): participants receiving sleep hygiene interventions ($n=23$) had significant reduction in ADHD symptoms and a significant improvement in sleep quality (Peppers et al., 2016). Corkum et al. (2016) investigated the effect of a distance intervention for insomnia in children (aged 5–12 years) with ADHD, which included parents being mailed intervention manual, diaries for tracking sleep and intervention implementation, and a reward chart with stickers and 5 weekly telephone sessions for coaching in the sleep intervention steps. Results from this study indicated significant improvement in three areas of sleep onset latency, bedtime resistance and sleep duration in the treatment group ($n=30$) as assessed through parent rating and in the area of sleep onset latency (Corkum et al., 2016).

The effect of a 5-week elimination diet on physical and sleep complaints in children with ADHD (treatment group $n=15$) were investigated by Pelsser et al. (2010) who reported a 77% decrease in the number of physical and sleep complaints. In another study, the effect of tryptophan enriched cereal intake on sleep of children with ADHD ($n=6$), Cerebral Palsy ($n=9$) and Autism Spectrum disorder ($n=7$) was studied; actual sleep time, sleep efficiency and immobile time improved in children with ADHD as measured through wrist actigraphy (Galán et al., 2017). The effect of 10 weeks' treatment with a polyunsaturated acid mixture on children with ADHD's behavior ($n=78$, 40 in treatment group) showed significant improvement in quality of life, ability to concentrate, hemoglobin levels and sleep quality (Yehuda et al., 2011). Sonne et al. (2016), studied the effect of MOBERO, a smartphone-based system that assists families in establishing healthy morning and bedtime routines in a 2-week intervention with children with ADHD ($n=13$, 6–12 years). The intervention was associated with 8.3% improvement in sleep habits, including positive change in seven of the eight CSHQ subscales (bedtime resistance, sleep duration, sleep anxiety, night wakenings, parasomnias, sleep disordered breathing and daytime sleepiness) and 16.5% reduction in core ADHD symptoms (Sonne et al., 2016).

ADHD Pharmacotherapy Effects on Sleep

Studies reviewed for the impact of ADHD medication on sleep are detailed in Table 7. A focus group study with parents ($n=16$) of children with ADHD (3–12 years) found that parents commence and continue pharmacotherapy for their children due to improvements in academic performance and social interactions, and cease therapy after their children experienced side effects including appetite suppression,

Table 7. Summary of Forty Two Included Studies Focusing on Pharmacotherapy and Its Effect on Sleep in Children with ADHD.

		Participant characteristics						Summary of main results
Authors/ publication year	Study design	Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age	Primary measures, limitations and biases	Objective	Interventions/comparisons/ outcomes
Ahmed et al. (2017)	Qualitative analysis of focus group discussions.	16 parents of ADHD children	For parents: 56%/44%	All children with ADHD whose parents participated were on prescription medication (methylphenidate, dexamphetamine, or atomoxetine).	3–12 years (parent age: 32–55)	Focus group (3). Limitations: Study not designed to address specific hypothesis, but a qualitative investigation, small number of participants, from a specific geographic urban location.	NA	Parents elected to cease pharmacotherapy after reported side effects including appetite suppression, weight loss, and sleep disturbances.
Bock et al. (2016)	Survey	67 respondent pediatricians (39% reported prescribing medication for ADHD)	N/A	N/A	NA	26-item survey. Limitations: Cross-sectional 6-month recall design subject to recall bias, bias to report socially desirable information, small sample.	NA	89% and 66% of the clinician respondents frequently use pharmacotherapy to treat paediatric sleep problems; few (20%) have received any training in this area. Melatonin (73%), OTC antihistamines (41%), antidepressants (37%), and benzodiazepines (29%) were the most commonly recommended medications.
Efron et al. (2014)	Cross sectional	ADHD:257 (57 on sleep medication)	86%/14%	53 children with ADHD were on medication	5–13 years	CSHQ, ADHD Rating Scale IV), ADHD medication use, Anxiety Disorders Interview Schedule for Children/Parent version IV, parent mental health (Depression Anxiety Stress Scale), Sleep log. Limitations: Study unable to ascertain indication for medication prescription, no control group, sample had children recruited for sleep-related studies, with 62% having moderate/severe sleep problems by parent report.	This study aimed to describe sleep medication use, as well as associated child and family characteristics in school-aged children with ADHD.	Children using ADHD medication were three times ($p = .05$) more likely to use sleep medication than children not taking ADHD medication; while children with combined-type ADHD were 2.5 times ($p = .04$) more likely than children with inattentive-type ADHD to use sleep medication and children with co-occurring internalizing and externalizing concerns were two times ($p = .04$) more likely to use sleep medication.
Becker, Pfiffner, et al. (2016)	Randomized Controlled trial	ADHD-In: 120, ADHD-Com.: 43	72%/28%	Stimulant naïve.	7–11 years	DISC-IV-Parent version, Vanderbilt ADHD parental version, Pittsburgh side effects rating scale. Limitations: Medication could not start from highest dose, or from placebo to highest dose, single parent-report item sleep measure used, results limited to initial medication titration, no examination of sleep during MPH maintenance or long-term; structure surrounding bedtime, parenting behaviors, or metabolism of MPH not explored, more ADHD-I children.	4-week, randomized, double-blind, placebo-controlled trial of once-daily (long-acting) methylphenidate (MPH)'s effect on predictors of sleep problems and other side effects.	Rates of reported sleep problems during the titration were 8.0% on placebo (7.4% moderate, 0.6% severe), 17.8% on low MPH dose (14.7% moderate, 3.1% severe), 14.7% on medium MPH dose (10.4% moderate, 4.3% severe), and 24.6% on high MPH dose (16.0% moderate, 8.6% severe). Weight (BMI) was significantly negatively associated with sleep problem scores on the low dose (Effect size = .03), medium dose (Effect size = .03), and high dose (Effect size = .04).

(continued)

Table 7. (continued)

Authors/ publication year	Study design	Participant characteristics				Primary measures, limitations and biases	Objective	Summary of main results
		Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age			Interventions/comparisons/ outcomes
Childress et al. (2009)	Randomized controlled trial	ADHD: 253	64.4%/35.6%	Medication naïve (175) or not treated with MPH-related medication in last 1 month (74).	6–12years	Recording of AEs, serious adverse events, vital signs, body weight, electrocardiogram (ECG), physical examination, hematology parameters, blood chemistry, and urinalysis, Conner's ADHD rating scales-parents and teachers (CADS-P&P), Global Impressions Scale (CGI-S). Limitations: In this forced-dose titration study, some subjects may not have been treated with optimal dose of d-MPH XR, the short, 5-week, duration does not allow for extrapolation of findings to long-term ADHD outcomes.	To examine the efficacy of 5 weeks, three doses of (10, 20, or 30 mg, once daily) dexmethylphenidate hydrochloride (HCl) extended-release (d-MPH XR; Focalin-XR)	All three doses of d-MPH XR were significantly more effective than placebo in improving ADHD symptoms as confirmed by parent ($p < .001$ for all three d-MPH XR groups). Decreased appetite and insomnia were the most frequently reported AEs leading to discontinuation (1.1% in "all d-MPH XR" group).
Lee et al. (2011)	Randomized controlled trial	ADHD: 157	All respondents were mothers	Previously prescribed MPH.	6–12years	Conner's Global Index for parents (CGI-Parents) and teachers (CGI-Teachers), Barkley Side Effects Rating Scale (SERS). Limitations: 0.5 mg/kg MPH for a 1-week period deviates from common clinical practice, use of only parent report for side-effects data. parents' treatment expectations/presence of parental ADHD not evaluated, majority of evaluations by mothers or both parents together, no data on teachers' gender preventing using gender as a covariate.	Random assignment, either placebo or 0.5 mg/kg/day MPH's efficacy for 1 week and the correlations of the side effects.	The greater "mood/anxiety" side effects on methylphenidate and placebo, the less the parents observe improvement of their children while treated with methylphenidate. Significant negative correlations were observed between the CGI-P response to MPH and the SERS parent ratings (placebo - MPH) for *irritability, prone to crying*, and *anxiousness* ($p < .005$). The poorer the therapeutic response, the higher these side effects were.
Lee et al. (2012)	Randomized controlled trial	ADHD:93	63 were eligible out of which (88%/12%)	Participants were not on medication 4 weeks prior to the study	6–12years	Clinical Global Impression of Severity (CGI-S) and the Clinical Global Impression-Improvement scale (CGI-I), Adverse events (AEs) chart, Sleep diary. Limitations: Study used sleep diary rather than objective measures, study used flexible titration method and did not divide the subjects into parallel-groups as per dose of MPH/age, lack of blinding, more than 30% of subjects did not complete procedure.	To study the effect of MPH (extended release and immediate release preparations) on sleep parameters.	MPH had negative impacts on sleep among young ADHD children (children between 6-years showed a 30 minutes decrease in total sleep time (TST) in the fourth week compared to baseline, Effect size: .22), but different preparations and doses did not affect the result (after baseline, there was an 11–21-minute decrease in TST for the lower dose group and a 16–25-minute decrease in TST for the higher dose group ($p < .05$, Effect Size: .10). There were no significant differences in TST changes between the lower and higher dose groups).

(continued)

Table 7. (continued)

Authors/ publication year	Study design	Participant characteristics				Primary measures, limitations and biases	Objective	Summary of main results
		Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age			Interventions/comparisons/ outcomes
Cockcroft et al. (2009)	Cross sectional	ADHD:23	69%/31%	(12 were on MPH, 11 medication naïve).	6.4–12.7 years	Parental questionnaire and the Wits Faces Sleepiness Scale. Limitations: Small sample size, no objective measure of sleep was used, no TD control group to assess if absence of pathology can also include daytime sleepiness.	To investigate whether treatment with methylphenidate had an effect on daytime sleepiness in children with ADHD and whether these changes could be noticed by parents and/or the children themselves.	There was a significant increase in perceived daytime sleepiness in the medicated group ($p < .05$), but not in the un-medicated group, between 08:30 and 13:00. The parents of the un-medicated group perceived their children as having significantly higher levels of daytime sleepiness between the hours of 13:00 and 15:00 ($p < .05$) than the parents of the medicated group.
Mohammadpour et al. (2018)	Randomized control trial	ADHD: 62	74%/26%	The participants were not on psycho-stimulants until 4 weeks before the testing.	5–12 years	Conner's Parent Rating Scale-Revised, ADHD rating scale-IV, Weekly Parent Ratings of Evening and Morning Behavior (WPREMB), Serum levels, Anthropometric variables, dietary intake, physical activity, sun exposure, and side effects. Limitations: Low doses of vitamin D and short duration of supplementation.	The effect of two groups receiving either 2000IU vitamin D or placebo in addition to MPH for 8 weeks.	Evening symptoms and total score of WPREMB scale were significantly different at weeks 4 and 8 between the two groups8 ($p = .013$, .016, respectively), but no differences were found in symptoms by CPRS and ADHD-RS scales, however ADHD-RS total score showed significant differences between week 4 and 8 only within vitamin D group ($p = .040$).
Mohammadzadeh et al. (2019)	Clinical drug trial	ADHD:66	74%/26%	Not mentioned.	6–12 years	ADHD parents rating scale, demographic checklist, drug complication checklist. Limitations: Small sample size, gender imbalance, no measurement of blood levels of EPA and docosahexaenoic acid, duration of the study was 8 weeks, which was a short time.	To study the efficacy between MPH with omega-3 group and MPH with placebo for 8 weeks.	The results showed no significant difference between the MPH with omega-3 group and MPH with placebo group based on the Parents ADHD Rating Scale between week 0 and week 8 ($p = .692$), Inattention ($p \geq .48$) and hyperactivity/impulsivity ($p \geq .80$) subscale scores.
Sonuga-Barke et al. (2009)	Randomized control trial	ADHD:184	74%/26%	99 children were on Concerta three times a day and 42 were on Equasym XL/Metadate CD two times a day.	6–12 years	Barkley Stimulant Side Effect Rating Scale (BSSERS). Limitations: Participants already being successfully treated with MPH so severe adverse events unlikely to be seen in study, trial underpowered for detecting rare events that could be severe, adverse events, measures of adverse events were derived only from parent ratings and not from direct observations of behavior.	Equasym XL/Metadate CD, Concerta, and placebo (PLA).	Treatment effects were seen only for emotionality (which improved $p = .002$) and sleep and appetite (which worsened ($p = .001$).

(continued)

Table 7. (continued)

Authors/ publication year	Study design	Participant characteristics				Primary measures, limitations and biases	Objective	Summary of main results
		Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age			Interventions/comparisons/ outcomes
Zarinara et al. (2010)	Randomized controlled trial	ADHD: 38 (MPH:19, Venlafaxine: 19)	71%/29%	Not mentioned.	6–12years	ADHD teacher and parent rating, complications and side effects record. Limitations: Lack of placebo group, use of only ADHD Rating Scale for measuring outcome, relatively small number of participants.	Randomly assigned to receive capsules of venlafaxine at doses of 50–75 mg/day or methylphenidate at a dose of 20–30 mg/ day for a 6-week.	Venlafaxine comparable to methylphenidate, improved symptoms of ADHD (significant effect of both protocols on Parent ADHD Rating Scale scores ($p < .001$), differences between two protocols were not significant at the endpoint ($p = .17$) but venlafaxine tolerability was superior with less headaches (57.89% vs. 15.78%, $p = .05$) and insomnia (52.63% vs. 10.52%, $p = .01$).
Ironside et al. (2010)	Placebo controlled medication trial	ADHD:16	75%/25%	Medication naïve.	6–12years	Actigraphy, Sleep diary. Limitations: Small sample size, medication administered at 4:00pm afternoon, failed to metabolize by night causing sleep onset problems, this could be explored by having varying timings for drug administration.	To examine the effect of 3-week MPH-Immediate Release trial on 24hour motor activity profile.	The children demonstrated significant increases in motor activity during the sleep-onset latency period (Placebo-23.63 (9.25), low dose MPH- 37.23 (11.11), High dose MPH- 37.60 (12.62), $p < .001$) and significant reduction in relative circadian amplitude (from a relative amplitude of 92.55 during the placebo condition to 84.16 on the low dose and 84.47 on the moderate dose ($p = .002$) and a phase-delay in the timing of the daily rhythm ($p = .03$).
Buchhorn et al. (2012)	Case control	ADHD: 23, Control: 19	82%/18%	11 children with ADHD received MPH medication	8–12years	Electro-cardiogram recordings. Limitations: Retrospective study, (preselection could imply that sample represents a risk subsample of children with ADHD). Additionally, uniform ADHD diagnostic study manual not applied, small group sizes.	Comparing the effect of MPH on Heart Rate Variability.	Compared to healthy controls, the ADHD children with and without MPH treatment showed significantly higher mean heart rates (ADHD without MPH: 94.3 ± 2.2; ADHD with MPH: 90.5 ± 1.8, controls: 84.7 ± 1.8, both ts >2.2, ps $< .033$), while the ADHD groups (MPH & no medication) ($t(29) = 1.7$, $p = .107$).

(continued)

Table 7. (continued)

Authors/ publication year	Study design	Participant characteristics: Sample size and diagnosis	Participant characteristics: Gender (male%/ female%)	Participant characteristics: Medication use	Participant characteristics: Age	Primary measures, limitations and biases	Objective	Summary of main results: Interventions/comparisons/ outcomes
*Wang et al. (2017)	Cross sectional	ADHD: 50, Control: 50	80%/20%	Not currently medicated.	6–12 years	SNAP-IV, CBCL, CPT, ADHD-RT, Saliva Cortisol test. Limitations: Saliva samples collected from patients in hospital but from healthy controls in school, no measurements of cortisol increment levels were made after baseline and during first month of treatment, waking time of participants was not precisely identified, patients with ODD or conduct disorder excluded.	To determine the trend in cortisol levels in children with ADHD and controls.	The cortisol levels of ADHD patients increased significantly after 1 month of MPH treatment (significantly higher than those at pre-treatment; mean difference = .11, $p = .046$). before decreasing to an intermediate level, but were significantly positively correlated with neuropsychological performance (salivary Cortisol was found to be independently and significantly correlated with impulse control ($\beta = -.006$, $p = .003$).
Slama et al. (2015)	Randomized controlled trial.	ADHD:36 (equally distributed between groups (placebo and osmotic-release oral system methylphenidate), Control: 40	100%/0%	The usual medication was replaced by OROS-MPH or placebo for 3 days and the subjects were tested on the third day, 8 to 10 hours after intake (at the end of the afternoon).	7–12 years	Continuous Performance Test-X [CPT-X], continuous performance test-AX [CPT-AX]), counting Stroop.Limitations: Study did not use computerized Stroop task, performance might have been influenced by presence of stimuli in area surrounding target, rating by observer may also result in relatively inaccurate scoring, only boys were tested.	A double-blind, randomized, placebo-controlled study investigating effect of OROS MPH.	Participants responded faster in CPT-AX (542 ms) than in the CPT-X (654 ms), $p < .001$, Reaction time latencies were influenced by treatment, $p = .026$ and ADHD children with OROS-MPH responded faster (559 ms) than ADHD children with placebo (638 ms, $p = .019$).
Faraone et al. (2009)	Randomized controlled trial.	ADHD:268 (81 received placebo, 98 received MTS, and 89 received OROS)	Not mentioned.	Participants abstained from any CNS active medication for 30 days prior to the study.	6–12 years	CSHQ, K–SADS–PL, K-BIT (intelligence). Limitations: For CSHQ 95th percentile and above considered to be of value 65/ above, and only 1 participant got that score (with the highest MPH dose), which might not be actual representation of high, rating scales of sleep problems may lack validity as compared with findings from sleep laboratories (using PSG, Actigraphy).	To examine indices of sleep behavior (including severity and frequency of each type of sleep problem) among ADHD children treated with either once-daily oral methylphenidate (osmotic-release oral system, OROS), the methylphenidate transdermal system (MTS), or placebo.	The main predictor of sleep problems was severity of pre-existing sleep problems ($\beta = .267$; $z = 9.68$; $p < .001$), whereas no significant linear effect of methylphenidate dosage was observed overall ($p = .135$), and this lack of effect was uniform across the two different methylphenidate preparations (OROS & MTS) as evidenced by the non-significant interaction of dose and treatment ($p = .852$).

(continued)

Table 7. (continued)

Authors/ publication year	Study design	Participant characteristics				Primary measures, limitations and biases	Objective	Summary of main results
		Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age			Interventions/comparisons/ outcomes
Ashkenasi (2011)	Retrospective randomized trial	ADHD: 26	73%/27%	All participants were allowed to be on study medication only.	6–12years	Sleep diary, Attention Deficit Hyperactivity Disorder Rating Scale-IV (ADHD-RS) and Connor's Global Impression-Parent (CGI-PS). Limitations: Small sample size did not permit closely examining individual signs from ADHD-RS-IV/CGI-Parent Scale (whether longer patch times help with symptom), reliance on caregiver reports of patch wear times and sleep parameters.	To study the effect of ransdermal MPH patch wear time (9hours, 10hours, 11hours, and 12hours) and randomization day (Monday, Tuesday, Wednesday, and Thursday) on sleep parameters.	Sleep parameters were not adversely affected by longer methylphenidate transdermal system patch wear times sleep latency ($p = .558$) or total sleep time ($p = .382$), however marginally significant trend toward better sleep quality at longer patch wear times ($F(1,341) = 3.60$, $p = .059$) was observed.
Findling et al. (2009)	Randomized controlled trial	ADHD: 327	64%/36%	Participants were not any (non-study related) medication during the study.	6–12years	Adverse events (AEs), physical examinations, vital signs, electrocardiograms, laboratory tests, the Children's Sleep Habits Questionnaire, and the occurrence of application-site reactions. Limitations: MTS tolerability/effectiveness assessed using open-label design, lacking blinding, susceptible to observer bias, lack of a placebo arm, results of long-term, open-label studies may be biased, as subjects remaining in study may continue to have improvements in ADHD symptoms, majority of participants were white males.	To assess the 12-month tolerability of MTS (MPH transdermal system) in children with ADHD.	81.3% reported adverse effects, of which 98.3% were mild or moderate in severity. Long-term tolerability of transdermal MPH found no apparent overall effect on sleep behavior, however the participants 8.9% incidence of Insomnia.
Chin et al. (2018)	Case control study	ADHD: 71, Control: 30	76%/24%	Participants were not medicated for ADHD in the past 6months.	6–12years	Polysomnography (PSG), Paediatric Sleep Questionnaire (PSQ). Limitations: One-night PSG at baseline and at 6months follow up, possible variability in measurements from night to night.	To study the effect of MPH treatment on sleep problems.	For the ADHD group after 6months' treatment PSG data showed significantly increased total sleep time ($p = .005$) and decreased periodic limb movement index (PLMI) ($p = .031$) after 6-month MPH treatment, significant increases in AHI ($p = .012$) and hypopnea counts ($p = .008$). For the PSQ data, significantly decreased rates of PLMD ($p = .029$) and sleep onset latency ($p = .021$) were shown.

(continued)

Table 7. (continued)

Authors/ publication year	Study design	Participant characteristics				Primary measures, limitations and biases	Objective	Summary of main results
		Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age			Interventions/comparisons/ outcomes
Morash-Conway et al. (2017)	Placebo controlled randomized trial	ADHD:21	80%/20%	Medication naïve.	6–12 years	PSG and cognitive assessment. Limitations: Need for larger/ varied sample size would allow more in-depth analyses, computerized test used (might not represent actual daily functioning), so classroom observation would have been apt, cognitive processes related to long-term academic and functional outcomes not explored.	4-week blinded placebo-controlled randomized trial of long-acting MPH.	Long-acting stimulant medication was found to be an effective treatment for enhancing alerting attention ($p = .003$ with tone), ($p < .001$, without tone) executive attention ($p < .001$), working memory ($p = .032$), and academic productivity ($p = .033$), but resulted in poorer sleep (children were sleeping on average 40 minutes less while taking medication).
Ricketts et al. (2018)	Cross sectional intervention study	ADHD:576 (Behavioral treatment ($n = 144$), Medication management ($n = 143$) Combined treatment ($n = 144$) Community care ($n = 145$).	80%/20%	175 on stimulant medication at study onset	7–9 years	SNAP-IV, CBCL, Children's Depression Inventory (CDI), Multidimensional Anxiety Scale for Children (MASC). Limitations: Sleep-related items in CBCL used do not compose well-validated sleep measure, homogenous sample (ADHD subtype- ADHD-Combined type), age range restricted to young children limiting extension of conclusions to older children/ adolescents, lower average daily MPH dose in community care may have confounded group effects.	The effect of MPH, behavior therapy and their combination, community care on sleep measures.	There was no significant effects of treatment assignment on baseline sleep problems score for medication ($z = -.37$, $p = .71$), behavioral treatment ($z = -.74$, $p = .46$), or combined treatment ($z = .44$, $p = .85$) relative to community care and also no significant simple effects of treatment assignment on posttreatment sleep problems score for medication ($z = -1.11$, $p = .27$) or behavioral treatment ($z = -1.17$, $p = .24$) relative to community care, however, combined treatment on sleep problems relative to community care ($z = -3.02$, $p = .003$).
Kim (2010)	Cross sectional intervention study	ADHD: 24	91%/9%	Stimulant naïve.	6–12 years	PSG, CSHQ, Barkley Adverse Effects Ratings Scale, CBCL, Clinical Global Impression (CGI) scale- Improvement, State Trait Anxiety Inventory, Yale Global Tick Severity Scale, K-ARS, K-SADS-PL-Korean version, Children's Depression Inventory (CDI). Limitations: Small sample size, open-label study (OROS MPH effectiveness could be overestimated and evaluation of adverse effects and tolerability limited by not having placebo comparator.	To study the effect of MPH-OROS extended release on sleep architecture measured through PSG & CSHQ.	After OROS MPH administration, percentage of stage 2 sleep was increased ($p = .024$), number of awakenings was decreased ($p = .047$) and relative to baseline, parasomnias scale's score on the CSHQ were decreased ($p = .033$), Sleep Onset. Latency was increased in children with subjective sleep difficulties (Effect size = .226). Bedtime Resistance and Sleep Onset Delay in Children's Sleep Habits Questionnaire were also increased for OROS MPH in individuals with sleep complaints (Effect size = .185; Effect size = .248 respectively).

(continued)

Table 7. (continued)

Authors/ publication year	Study design	Participant characteristics				Primary measures, limitations and biases	Objective	Summary of main results
		Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age			Interventions/comparisons/ outcomes
Kim et al. (2011)	Cross sectional intervention study	ADHD: 102	92%/82%	All participants were on a daily dose of MPH for 4 weeks, and stable dose of MPH for 3 weeks before the study.	6–12 years	Inattentive/Overactive and Oppositional/Defiant subscales of the IOWA Conner's Rating Scale, Clinical Global Impression (CGI) scale, parents/caregivers rated sleep quality using a 4-point scale (poor, fair, good, excellent) during all visits. Limitations: Switching process resulted higher daily MPH doses although differences were not large, groups receiving twice a day vs. three times a day MPH-IR regimens not separately compared, investigators-participants not blind treatment conditions, no control group.	To evaluate the efficacy and safety of OROS-MPH among children with ADHD who had been previously treated MPH-IR.	Parent/caregiver ratings on the Conners subscale, showed statistically significant improvement after 4 weeks of treatment with OROS-MPH ($p < .001$). However, the teachers' ratings on the same subscale did not reflect any significant improvemen There were no statistically significant changes in sleep related adverse effects for both preparations of MPH.
Pelham et al. (2011)	Randomized controlled trial	ADHD: 9	100%/0%	All participants were receiving a stable dose of IR MPH before enrolment, but none had previously been treated with Extended release stimulants.	6–9 years	Twelve hourly assessments of classroom behavior and productivity were completed with efficacy measures of rule violations, math correct, Inattention/over activity teacher rating/oppositional defiant teacher rating. Limitations: Small sample size, all male, 80% comorbid ODD/CD, MTS applied for 24 consecutive hour exceeding FDA-approved wear time of 9 hour, all participants previously stabilized on MPH (cannot generalize the tolerability findings to stimulant naïve children).	Efficacy and tolerability of MPH- transdermal formulation (MTS) against Immediate Release- MPH (IR-MPH) and placebo in a 12-hour analog classroom setting.	MTS demonstrates comparable efficacy (for rule violations ($p = .01$), math correct responses ($p < .001$, only for MPH), inattention ($p = .02$)and oppositional defiance ($p = .02$) ratings) and tolerability (non-significant differences in adverse reactions) to TID IR MPH.

(continued)

Table 7. (continued)

Authors/ publication year	Study design	Participant characteristics: Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age	Primary measures, limitations and biases	Objective	Summary of main results: Interventions/comparisons/ outcomes
Pliszka et al. (2017)	Randomized controlled trial	ADHD: 163 (Delayed Release DR/Extended Release ER-MPH, $n=81$; placebo, $n=80$)	70.2%/29.8%	Stimulants, clonidine, and guanfacine required a ≥72-hour washout, and any other medication to treat ADHD required a ≥7-day washout before randomization.	6–12 years	ADHD rating scale-IV (ADHD-RS-IV), Before-School Functioning Questionnaire (BSFQ), Parent Rating of Evening and Morning Behavior-Revised, morning (PREMB-R AM) and evening (PREMB-R PM). Limitations: Short study duration limits ability to extrapolate findings over long term, participants aged 6–12 years, therefore, applicability of findings to other age groups unknown, enrolled participants previously shown partial response to MPH (therefore, response and safety profiles in MPH-naïve patients may be different than those achieved in this study).	3-week trial of delayed-release and extended-release methylphenidate (DR/ER-MPH) formulation.	DR/ER-MPH was generally well tolerated and demonstrated significant improvements versus placebo in ADHD symptoms (after 1 ($p<.001$) and 2 weeks of treatment ($p=.002$) and at-home functional impairments (early morning, late afternoon and evening) (PREMB-R, $p<.001$) and PREMB-R PM ($p=.002$).
Giblin and Strobel (2011)	Randomized controlled trial	ADHD: 24 (3, 11, 2, 8 for the 30 mg/d, 50 mg/d, 70 mg/d, and placebo groups, respectively)	41.6%/58.4%	21 children had previously been on medication.	6–12 years	PSG, Actigraphy, CSHQ, Sleep hygiene/ sleep schedule inctructions, ADHD-RS IV, Clinical Global Impression-Severity Scale (CGI-Impressions), Conner's Parent Rating Scale-Revised: Short Form. Limitations: Effect of LDX treatment assessed in relatively small patient sample, comorbid psychiatric diseases excluded, limiting generalization of results to broader population of ADHD, PSG data may in part be explained by habituation sleep laboratory setting.	3 doses of Lisdexamfetamine Dimesylate (LDX) (30, 50, and 70 mg/ day) over a 10-week period	Number of awakenings as measured by PSG data was significantly decreased from 7.9 ± 4.5 to 3.3 ± 4.3 in the LDX-treated group when compared to baseline ($p<.0001$), however no difference was seen for total sleep time or Wake after sleep onset measures. No significant difference was seen for actigraph data or CSHQ reports.
Wigal et al. (2009)	Randomized controlled trial	ADHD; 129 (30 mg/d- 58, 50 mg/d- 50, 70 mg/d- 21)	76%/24%	Washout period of 7 days before start of study for participants if applicable	6–12 years	SKAMP-D, Permanent Product Measure of Performance (PERMP), CGI, ADHD-RS-IV, blood pressure, ECG, clinical laboratory test, treatment emergent adverse effects screening. Limitations: Short treatment duration and assessment phases may underestimate TEAEs, subjects with severe comorbid psychiatric conditions excluded, unblinded design- thereby subject to biases, no measurements beyond 13 hours post dose captured, lack of subjects with inattentive type ADHD, under-representation of minority populations.	4-week dose-optimization of LDX (30, 50, 70 mg/day	Treatment emergent adverse effect (insomnia-27%).

(continued)

Table 7. (continued)

Authors/ publication year	Study design	Participant characteristics				Primary measures, limitations and biases	Objective	Summary of main results
		Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age			Interventions/comparisons/ outcomes
Wigal et al. (2012)	Intervention study	ADHD: 27	77.8%/22.2%	Stimulant washout period of 7 days before start of study for participants (14) if applicable	6–12 years	CGI, ADHD-RS-IV, treatment emergent adverse effects screening. Limitations: Small sample size, few female subjects, no randomization to placebo within exposure based groups to allow comparison to potential placebo effects, no inclusion of untreated group.	LDX, initiated at 30 mg, was dose titrated in 20 mg increments to a possible 70 mg over 4–5 weeks, and its effect was studied on the initial onset of efficacy when compared to placebo.	The stimulant naïve group as well as the previous-exposure group reported trouble sleeping and stomach pain with a greater incidence for stimulant naïve subjects ($p = .004$, and $p = .0034$, respectively), hyperfocus ($p = .025$) was only seen in the stimulant naive subjects, however only previous-exposure subjects experienced dizziness ($p = .037$).
Rugino (2018)	Randomized controlled trial	ADHD: 29	58%/42%	12 participants were on stimulant medication before beginning of study, and 6 out of them had a stimulant washout period before commencing study.	6–12 years	Polysomno-gram, a CSHQ, an ADHD-RS, ADHD CGI-I, a sleep CGI-I, vital signs, growth parameters, physical examination, detailed interval medical history, ECG, and laboratory investigations. Limitations: Small sample size, short treatment time, single PSG may not represent effect on sleep at home over extended time, data cannot be generalized to children administered GXR in the evening, cannot be generalized to ADHD population without primary sleep disorder, unequal sample sizes at baseline and termination not explored.	Guanfacine extended release (GXR) administration.	Although none of the PSG measured sleep parameters demonstrated difference between GXR and placebo, ADHD Rating Scale-IV combined scores for guanfacine extended release and placebo groups at baseline and at endpoint showed difference ($p < .001$, effect size: .41).
Young et al. (2014)	Randomized controlled trial	ADHD: 333 (GXR a.m., $n = 107$; GXR p.m., $n = 114$; placebo, $n = 112$)	70%30%	Not mentioned.	6–12 years	Paediatric Daytime Sleepiness Scale (PDSS), Conner's Parents Rating Scale – Revised (CPRS-R), Adverse effects, laboratory tests, physical examinations. Limitations: CPRS–R:S modified to evaluate ADHD-symptoms at several time points throughout the day, which may limit interpretation results, study was not powered to formally assess differences between the a.m. and p.m. cohorts, the PDSS has not demonstrated correlation with objective measures such as PSG, as a self-report measure, results may be subject to rater bias.	Once-daily Guanfacine extended release (GXR) monotherapy administered either in the morning or evening.	Subjects receiving GXR showed significantly greater improvements from baseline compared with placebo, regardless of time of administration ($p < .003$ vs. placebo across all subscales for GXR a.m. and GXR p.m.) Effect sizes were similar for both morning and evening GXR administration (.71 and .62, respectively). The most frequently reported AEs (reported in >10% of subjects) in the GXR groups were somnolence, headache, sedation, upper abdominal pain, and fatigue.

(continued)

Table 7. (continued)

Authors/ publication year	Study design	Participant characteristics				Primary measures, limitations and biases	Objective	Summary of main results
		Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age			Interventions/comparisons/ outcomes
Connor et al. (2010)	Randomized controlled trial	ADHD: 217	68.7%/38.3%	Participants were not on any medication.	6–12years	Conner's Parents Rating Scale – Revised (CPRS-R), ADHD-RS-IV, ECG and laboratory tests. Limitations: Subjects co-existing psychiatric conditions excluded, study duration only 8weeks, study did not include teacher ratings to assess behavior in classroom, study not designed to investigate effects of guanfacine XR on oppositional symptoms.	Guanfacine extended release (1–4mg/ day) or placebo for 9weeks to examine its safety and efficacy outcome.	Reduction in ADHD-RS-IV total score from baseline to endpoint in Guanfacine-treated group compared with the placebo group (23.8 vs. 11.5, respectively; $p < .001$; effect size = .92), percentage reduction from baseline to endpoint in CPRS-scores for oppositional subscale and ADHD-RS-IV total scores indicated that the decrease between the two was highly correlated ($r = .74$). Most commonly reported, treatment-emergent AEs in the guanfacine XR group was somnolence (50.7%).
Montoya et al. (2009)	Randomized controlled trial	ADHD: 113 (children group)	79.5%/20.5%	Medication naïve.	6–12years	Conner's Parents Rating Scale – Revised (CPRS-R), ADHD-RS-IV, ADHD Clinical Global Impression on severity (CGI-ADHD-S). Limitations: Small sample size (less statistical power), due to short duration, effect size on symptoms ratings beyond week 12, the development of tolerance, the occurrence of hepatic disorders or long-term consequences of vital signs changes, could not be addressed, teacher ratings not obtained, neuropsychological tests not included.	Atomoxetine or placebo, respectively, for 12weeks to examine its safety and efficacy.	Treatment-related adverse events were significantly more frequent with atomoxetine (65.0%) than with placebo (37.3%), the most frequent being decreased appetite ($p = .006$) and somnolence ($p = .002$).
Wehmeier et al. (2011)	Randomized controlled trial	ADHD: 105 (ATX group: $n = 54$; placebo group: $n = 51$)	77.6%/32.4%	Atomoxetine naïve, not currently treated with other psychotropic drugs.	6–12years	Computerized performance test (CPT), infra-red motion tracking device, Weekly Ratings of Morning. Limitations: Relatively short duration of observation period, CPT tool not gold standard tool for assessing ADHD symptom severity, be the close oversight of the patient bythe physician may have influenced Clinical Rating Scale scores.	Atomoxetine (ATX) dose of 1.2mg/kg/day for 8weeks' effect on neuro-cognitive performance.	The WREMB-R total score (effect size − 1.00) and the sub scores (late noon and evening subscore-effect size 1.02 & Difficulty falling asleep- effect size- .62) showed statistically significant differences between the treatment groups at week 8 ($p < .001$) ATX was significantly superior to placebo in reducing ADHD symptom severity as measured by ADHD-RS (effect size- 1.30) and CGI-S scores (effect size − 1.11).

(continued)

Table 7. (continued)

Authors/ publication year	Study design	Participant characteristics				Primary measures, limitations and biases	Objective	Summary of main results
		Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age			Interventions/comparisons/ outcomes
England et al. (2011)	Randomized controlled trial	ADHD, ADHD+Restless Leg syndrome: 29	68%/32%	All participants were CNS active medication naïve.	7–12 years	PSG, Conner's rating scales and neuro-psychometric testing at baseline and endpoint. Limitations: Study did not directly test impact of improvement of RLS symptoms on quality of life, small sample size.	Carbidopa/L-DOPA or placebo for 8 to 13 weeks to see if ADHD symptoms improve differentially in children with and without RLS/PLMS.	ADHD was more severe in children without RLS/PLMS than in children with RLS/PLMS ($p = .006$), however L-Dopa significantly improved RLS/PLMS ($p = .007$) but not ADHD.
Ferri et al. (2013)	Case control study with second phase of randomized controlled trial with ADHD patients	ADHD: 18, Control: 17	61%/49%	Participants were not on medication.	7–12 years	PSG, scoring of leg movements. Limitations: Lack of comparison group (children with RLS but not ADHD); no data on iron status, results more relevant (children with ADHD accompanied by sleep disturbance than for ADHD in general).	To assess the effect of Carbidopa 25 mg/L-DOPA 100 mg CR per tablet on changes on the leg movement (LMs) time structure in ADHD children.	LMs during sleep in children with ADHD do not show a highly periodic character and are not considerably modified by *L*-DOPA treatment.
Hoebert et al. (2009)	Randomized controlled trial	ADHD+Sleep Onset Insomnia: 93	74.5%/26.5%	Participants were not on stimulant medication at the beginning of the study. Information about other medication use not mentioned.	6–12 years	Parent questionnaire with combination of multiple choice, numeric, open ended and scaled questions, 19 in total. Limitations: Parents reporting adverse events from Melatonin not carefully interviewed, co-medication not reported, lack of measures to assess long-term effects of melatonin treatment on pubertal development and fertility, lack of control treatment.	Longitudinal follow up (average 3.7 years) study of Melatonin use and discontinuing.	Melatonin is an effective therapy for CSOI in children with ADHD, but does not reduce ADHD symptoms themselves 67 children temporarily discontinued Melatonin and 22% discontinued completely. For those discontinued completely- Dim Light Melatonin Onset (DLMO) for the discontinuing children when compared to rest of subjects was not different ($p = .413$, effect size = .09). For those who discontinued temporarily- effects in: No change of sleeping pattern (1.5%). Delay of sleep onset time (92.3%). Delay of wake up time (30.8%). Changing daytime behavior (29.2%) was observed when compared to the rest of the subjects, who did not discontinue ($p = .06$).

(continued)

Table 7. (continued)

Authors/ publication year	Study design	Participant characteristics				Primary measures, limitations and biases	Objective	Summary of main results
		Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age			Interventions/comparisons/ outcomes
Masi et al. (2019)	Naturalistic treatment with MPH	ADHD: 42 children under 12years	93%/7%	Participants were on MPH medication.	6–12years	K-SADS-PL, Clinical Global Impression Severity (CGI-S), Conner's Parents and teacher rating scale. Limitations: CGI-I used as outcome measure, not measure of sleep disorder severity and improvement, lack of previously validated questionnaires, sleep diary and actigraphy, limits reliability of results, lack of specific information about sleep habits before starting MPH.	MPH mean dosage average 33.5 mg/ day, melatonin on sleep (mean dosage average 1.85 mg/d) for 4 weeks, to gage effectiveness.	Melatonin is effective (clinical severity for the CGI-S reduced to 2.13 ± 1.05 at follow up from $3.41 \pm .70$ at baseline ($p < .001$) treatment, irrespective of gender, age and comorbidities, for ADHD children with sleep problems.
Blumer et al. (2009)	Randomized controlled trial	ADHD: 111 (6–11 years)	Not mentioned.	Nonhypnotic medications were used for sleep by 41 patients in the zolpidem group (Clonidine) and 23 patients in the placebo group (Antihistamines and other drugs with drowsiness as a side effect).	6–11years	PSG, Clinical Global Impression scale (CGI), Peadiatric Daytime Sleepiness Scale (PDSS), ADHD-RS-IV, Conner's Continuous performance test- II (CPT-II), adverse effects assessment. Limitations: Potential for drug interactions not noted, responses to zolpidem for children receiving different medications for insomnia and/ or ADHD before the study were not evaluated.	To evaluate the hypnotic efficacy of Zolpidem 0.25 mg/ kg per day among ADHD children.	Zolpidem at a dose of 0.25 mg/ kg per day failed to reduce the latency to persistent sleep on PSG recordings (20.28 vs. 21.27 minutes).
Lyon et al. (2011)	Randomized controlled trial	ADHD:98	100%/0%	27 on stimulant medication during study (13-treatment group and 14-placebo group).	8–12years	Peadiatric Sleep Questionnaire (PSQ), Actigraph. Limitations: Only male participants, no other social-emotional functioning scale used, therefore behavioral outcomes not examined.	L- theanine 400 mg daily at breakfast and after school on sleep quality.	Increased percent of time spent in restful sleep in L-theanine compared to placebo group ($p < .05$), fewer bouts of nocturnal activity in L-theanine compared to placebo group, $p < .05$, lower number of minutes spent awake after onset of sleep in the L-theanine compared to the placebo group ($p < .058$).

(continued)

Table 7. (continued)

Authors/ publication year	Study design	Participant characteristics				Primary measures, limitations and biases	Objective	Summary of main results
		Sample size and diagnosis	Gender (male%/ female%)	Medication use	Age			Interventions/comparisons/ outcomes
Blader et al. (2009)	Randomized controlled trial	ADHD: 27	77%/23%	16 children were on medication at enrolment	6–13 years	Retrospective-Modified-Overt Aggression-Scale, Child-Behavior Ckecklist (CBCL), K-SADS-PL, Conner's Global Index- parent version, Barkley Behavior and Adverse Events Questionnaire–Modified. Limitations: Small sample size.	Titration began with triphasic-release MPH preparation (Concerta) 90 mg/ day or biphasically released MPH product (Metadate CD) daily dose 60 mg, concurrent psychosocial treatment-Community parent education program for 5 weeks, Divalproex extended-release once-daily preparation for 8 weeks to examine the effect of reducing aggressive behaviors.	The odds of remission with divalproex treatment were seven times greater than that with placebo, although a small sample size rendered the confidence interval (CI) for this estimate especially wide (odds ratio = 7.33, $p < .05$).
Newcorn et al. (2013)	Randomized controlled trial	ADHD: 332, GXR AM ($n = 107$), GXR PM ($n = 114$), or placebo ($n = 112$).	70%/30%	Not mentioned.	6–12 years	ADHD-RS-IV, Pediatric Daytime Sleepiness Scale, Medical Dictionary for Regulatory Activities (MedDRA). Limitations: Study was not adequately powered to detect small differences between GXR AM and GXR PM treatment arms, drug administration times limited to 2 time points: morning or evening, subjects not required to complete self-report or structured scale of AEs (caregiver ratings may be subject to underreporting, rater bias or halo effects), subject cohort consisted predominantly of white males.	8-week, double-blind, dose-optimization study for morning and evening Guanfacine administration.	Treatment-emergent adverse events were although mild or moderate in severity; the most common was somnolence (GXR all-active: 44.3%; GXR AM: 46.7%; GXR PM: 42.1%; placebo: 12.5%). Significant differences in ADHD-RS-IV total scores between GXR & placebo- Effect sizes were .77, .75, and .78 for the all-active, GXR AM, and GXR PM groups, respectively.

weight-loss and sleep disturbances (Ahmed et al., 2017). Bock et al. (2016) stated that clinicians ($n=67$) reported that 39% of patients with childhood ADHD seek over the counter or prescription medication for their sleep problems. Further sleep medication use (melatonin 14% and clonidine 9%) in 5 to 13-year-old children with ADHD ($n=257$) was associated with their intake of ADHD medication, combined ADHD subtype and internalizing/externalizing comorbidities (Efron et al., 2014).

In children with ADHD ($n=163$, 7–11 years), increased Methylphenidate (MPH) dose is associated with increased sleep problems, particularly for lower weight/BMI; 23% of children without pre-existing sleep problems were reported to have sleep problems at the highest MPH dose (Becker, Froehlich, et al., 2016). Childress et al. (2009) reported that psychiatric side effects, events including insomnia occurred more often in patients assigned to the higher doses of dex-MPH Immediate release (20, 30 mg/day) in a study including 253 children with ADHD between 6 and 12 years of age. For similar age group participants, Lee et al. (2012) reported MPH had a negative effect on later bedtimes, wake-up times and total sleep times for children with ADHD (TST was reduced by about 17 minutes on weekdays and 29 minutes at weekends, no difference between higher or lower doses, total $n=93$). Further, parent-reported side effect ratings ($n=157$) revealed significantly more insomnia in the MPH treatment group as compared to the placebo group (Lee et al., 2011). Further in another similar age group of children with ADHD on MPH ($n=12$) were reported to be significantly sleepier as the morning progressed, although at no point did they feel sleepier than 11 medication naive children (Cockcroft et al., 2009). When outcomes for MPH and Vitamin D supplement were assessed in children with ADHD ($n=25$, 5–12 years), improvements in parent ratings for evening behaviors including getting ready for bed and falling asleep were reported compared to a control group ($n=29$) receiving MPH and placebo (Mohammadpour et al., 2018). Omega-3 supplementation with MPH for children with ADHD ($n=33$, 6–12 years) revealed no significant sleep related drug complication differences when compared to MPH and placebo group ($n=33$; 2.9 &4 % respectively) (Mohammadzadeh et al., 2019). In a Venlafaxine versus MPH effect study, side effects of insomnia were more frequently observed in the MPH treatment group ($n=8$, 6–12 years) (Zarinara et al., 2010). Sonuga-Barke et al. (2009) showed that while sleep problems significantly increased as a function of MPH treatment among children with ADHD, children who showed sleep-related adverse events also showed appetite-related adverse-events as reported by parents (total $n=184$, 6–12 years).

Regarding MPH formulation, a handful of studies have examined the impact on sleep parameters. For extended release MPH, a trial with children with ADHD ($n=24$, 6–12 years) comparing polysomnography and CSHQ results before and after the treatment found that sleep onset latency did not change, but that the number of awakenings decreased, and the percentage of stage 2 sleep increased during treatment (Kim, 2010). Switching from MPH-Immediate release to MPH extended release revealed no changes in sleep in 102 six to twelve-year-old children with ADHD (Kim et al., 2011). An efficacy and tolerability assessment of MPH transdermal formulation versus MPH immediate release found that both medications were well tolerated with only mild reductions in sleep onset latency (Pelham et al., 2011). A placebo controlled 3-week trial for assessing MPH extended release(ER) and delayed release (HLD200) revealed insomnia (>10%) as a commonly reported treatment emergent adverse effect for both the 6 to 12 years aged treatment groups ($n=81$) as compared to placebo ($n=80$) (Pliszka et al., 2017).

Findings of adverse effects of MPH on sleep are not universal. Faraone et al. (2009) reported that neither once-daily oral ($n=89$) nor transdermal formulations ($n=98$) of methylphenidate reliably elicit sleep problems or influence the severity of such problems when they occur along with no significant effect of dose in 6 to 12 year olds with ADHD. On similar lines in the same age group, Ashkenasi (2011) reported that transdermal MPH patch wear times (9, 10, 11, and 12 hours per day) exerted no significant effect on sleep latency or total sleep time on children with ADHD ($n=26$). A study investigating long-term tolerability of transdermal MPH found no apparent overall effect on sleep behavior for the children ($n=327$, 6–12 years) as per parent reports on CSHQ (Findling et al., 2009). Chin et al. (2018) showed significantly increased total sleep time and reduced periodic limb movement index along with parent-reported reduction in bruxism and snoring in ADHD-Inattentive children ($n=35$), as well as nightmares in ADHD-Combined presentation ($n=36$) as an effect of MPH. Morash-Conway et al. (2017) report that following MPH treatment, sleep duration in children with ADHD ($n=21$) was positively related to performance improvement on an executive attention task. Ricketts et al. (2018) found no deleterious effect of MPH on sleep ($n=143$) and that combined treatment with stimulant medication plus behavior therapy ($n=144$) was associated with statistically significant reductions in sleep problems.

A number of studies have examined the effects of MPH on circadian function in children with ADHD. Ironside et al. (2010) reported that for children with ADHD on MPH, mean motor activity levels (Mesor) were elevated during the down interval time (the sleep-onset latency period after lights out when children were in bed trying to sleep; $n=16$, 6–12 years). A prospective 24-week observational study to assess trends in morning salivary cortisol levels among children with ADHD on MPH revealed that for these 6 to 12-year-old children ($n=50$) morning salivary cortisol level increased significantly during the first month of MPH treatment, before the level subsequently dropped to an intermediate level that

was not significantly different from either the baseline or 1-month level over the 6-month course of treatment (Wang et al., 2017). Slama et al. (2015) found a positive effect of MPH in CPT assessment for children with ADHD (total $n=36$, 7–12 years), with faster and less variable reaction times than under placebo during late afternoon, 8 hours after medication. Medication with MPH has also been reported to shift heart rate variability markers toward levels of normal controls (Buchhorn et al., 2013).

Three included studies examined the impacts of amphetamines on sleep measures in children with ADHD. Giblin and Strobel (2011) studied the effects of Lisdexamphetamine (LDX) on sleep in children with ADHD (treatment $n=16$, 6–12 years) using polysomnography, actigraphy and subjective sleep measures and report that LDX did not contribute to sleep disturbances as measured by both objective and subjective sleep parameters. However, 27% of children with ADHD treated with LDX ($n=129$, 6–12 years) experienced the treatment emergent side effect of insomnia (Wigal et al., 2009). On similar lines for the same age group, Wigal et al. (2012) found one of the significant side effects of "trouble sleeping" for LDX, although stimulant-naïve children ($n=13$) had significantly greater difficulty sleeping compared to those previously exposed to stimulants ($n=14$).

Examining the effects of non-stimulant ADHD medication on sleep parameters, Guanfacine treatment among 6 to 12 years' children, polysomnographic evaluation found that morning administration of Guanfacine extended release medication ($n=11$) had an untoward effect on the polysomnographic sleep outcome measure of total sleep time and total time in slow wave sleep (found to be shorter) compared with morning administration of placebo ($n=16$) (Rugino, 2018). For the same age group ADHD children, Guanfacine extended release use was not associated with subjective self-report of daytime sleepiness for morning ($n=107$) or the evening ($n=114$) administration groups (Young et al., 2014), although the same study reported somnolence as one of the frequent treatment emergent adverse effect. Somnolence was also reported as one of the primary adverse events associated with Guanfacine extended release treatment ($n=138$, 6–12 years) of children with ADHD by Connor et al. (2010). For treatment with atomoxetine, Montoya et al. (2009) demonstrated significantly more frequent adverse event of somnolence (24%) for atomoxetine compared to placebo (total $n=113$). However, a subjective ADHD-related morning and evening behavior rating scale (including an independent item: difficulty falling asleep) for 6 to 12 year olds children yielded improved scores for the treatment group ($n=54$) using Atomoxetine as compared to 51 receiving placebo (Wehmeier et al., 2011).

For other pharmacological treatments, one randomized controlled trial (RCT) reported that L-DOPA improved restless leg syndrome and periodic leg movement syndrome although it did not improve ADHD symptoms, sleep parameters, or neuro-psychometric measures in 7 to 12 year-old ADHD patients ($n=15$) when compared to 14 receiving placebo (England et al., 2011). However, within a similar age group, Ferri et al. (2013) found that Levodopa did not modify leg movement time structures for children with ADHD yet improved sleep latency (treatment $n=10$). Hoebert et al. (2009) reported melatonin treatment to be effective in 88% of cases of children with ADHD and chronic sleep onset insomnia ($n=105$, 6–12 years). Melatonin treatment in ADHD patients (children under 12 years $n=42$, 6–12 years) also treated with MPH and who developed sleep problems was found to be effective in improving sleep problems in 60.8% of the patients, with this efficacy was similar in males and females and in children when compared to adolescents (Masi et al., 2019). An 8-week, placebo controlled study to investigate the efficacy of zolpidem for the treatment of insomnia associated with ADHD revealed no significant change in sleep latency between baseline and week 4, and no significant difference in scores for the CGI (Clinical Global Impression) scale were observed between weeks 4 and 8 among the age group of 6 to 11 year children (treatment $n=136$) (Blumer et al., 2009). In an objective sleep quality study, actigraphy data indicated that ADHD boys (8–12 years) who consumed L-theanine ($n=46$) obtained significantly higher sleep percentage and sleep efficiency scores, along with a non-significant trend for less activity during sleep (defined as less time awake after sleep onset) compared to 47 of those in the placebo group (Lyon et al., 2011). Finally, Blader et al. (2009) reported that children with ADHD (6–13 years) whose disruptive psychosocial behaviors were unresponsive to stimulants and were treated with divalproex ($n=15$) tended to have higher rates of trouble falling asleep than children treated with placebo ($n=15$).

Discussion

The current review aimed at providing a comprehensive overview of the associations between sleep and circadian function with ADHD in children. The breadth of the topic and the need for the review was reflected in the large number of studies included.

For the subjective assessment of sleep in children with ADHD, 119 (out of the total 148) selected articles utilized subjective parental/caregiver reports of sleep, and these reports highlighted higher occurrence of bedtime resistance, sleep anxiety, sleep onset delay, sleep disordered breathing, day time sleepiness and a general trend of poorer sleep quality, duration and efficiency (Abou-Khadra et al., 2013; Akinci et al., 2015; Becker, Pfiffner, et al., 2016; Chiraphadhanakul et al., 2015; Gomes et al., 2014; Moreau et al., 2013; Scott et al., 2013). In assessing studies utilizing parental/caregiver reports of childrens' sleep, it is important to recognize various sources of potential bias in such reports and the bidirectional nature of the relationship between children's sleep and parental/caregiver wellbeing, and the consequent potential

for altered subjective appraisal of children's sleep parameters. The relationship between parental stress and sleep problems in children with ADHD wherein children's sleep dysfunction causes a decrease in the parent's wellbeing and increased fatigue was noted in the review of Martin et al. (2019), and such effects may decrease parents' ability to implement more consistent/ effective behavioral and sleep management strategies for the child. As such, an important limitation for some of the reviewed articles is the absence of information about parental stress, presence of family psychiatric history, family's social-economic status or parent's education (Bergwerff et al., 2016; Bériault et al., 2018; Eyuboglu and Eyuboglu, 2018; Kwon et al., 2014; Matsuoka et al., 2014; Williams et al., 2016). Studies included in the current review also indicate that sleep problems in ADHD are associated with externalizing and internalizing comorbidities in the child and caregivers' own attitudes, sleep quality, lifestyle trends and socio economic conditions (Bessey, Richards, et al., 2013; Lycett, Mensah, et al., 2014; Matsuoka et al., 2014; Sciberras et al., 2016, 2017). Further, it is clear that sleep in children with ADHD should be considered carefully to all aspects of the child's environment, and such relationships may be best studied through well-powered and carefully constructed longitudinal studies. Keeping in mind our selection criteria, we found 15 studies that have used a longitudinal design out of the total 148 reviewed articles. However longitudinal studies also reported specific weaknesses such as missing data, significant drop out rate for participants, all measures not assessed during baseline and follow-up consistently and data assessed through older or previous editions of measures (Mulraney et al., 2017; Schmid et al., 2014; Soehner et al., 2019). Additionally, given the fact that subjective ratings do not always bring about a comprehensive account of sleep related concerns among ADHD children, qualitative analysis of reports from caregivers/clinicians may be explored to examine sleep in childhood ADHD. As such, there are no studies that have explored sleep problems in ADHD children through qualitative methods such as thematic analysis.

Considering the nature of measures employed in studies based on subjective accounts, our review found wide ranging and dominant use of a few standard tools such as the Child Sleep Health Questionnaire (CSHQ), Pittsburgh Sleep Quality Questionnaire and Childhood Sleep Disturbance Scale (CSDS) etc. Although some of these scales have individual sub-scales and a global measure of sleep disturbance, how parents understand the difference between items while rating them and how they understand their child's specific sleep problems should be considered, as should the lack of congruence with results from objective measures such as actigraphy (Akinci et al., 2015; Choi et al., 2010). Further, sleep questionnaires do not usually assess information regarding restless leg syndromes or breathing problems during sleep, unless the tool is built for assessing such concerns specifically. Hence, to reach more consistent and comprehensive set of results, studies exploring similar variables through both objective and subjective measures is recommended. In our reviewed list of articles, there were 44 studies (out of the total 148) which utilized a combination of both subjectively reported and objectively measured sleep functions, thereby underlying the need for more such investigations.

We uncovered mixed evidence for greater subjectively-reported sleep problems in ADHD hyperactivity and combined presentation, than the inattentive subtype (Eyuboglu and Eyuboglu, 2017; Grünwald and Schlarb, 2017; Hansen et al., 2011; Sciberras et al., 2016). Similar links between sleep problems and hyperactivity or combined subtypes have been reported for older adolescents (Chiang et al., 2010; Mayes et al., 2009). Interestingly, among the reviewed studies, an important limitation is the lack of ADHD subtype-based recruitment of participants (for articles exploring the nature of sleep and its consequences for the ADHD child), leading to non-comparable number of children exhibiting ADHD-I, H or C presentations, thereby most studies generalizing the sleep problems for all subtypes. Further, Tsai et al. (2019) found that relative to inattentive subtype, childhood combined subtype was associated with higher risks of sleep disorders during adulthood.

Studies based on objective sleep measures reported shorter sleep duration, longer sleep latency, lower sleep efficiency, greater wakening after sleep onset and sleep fragmentation in ADHD (Lee et al., 2014; Miano et al., 2019; Moreau et al., 2013). However, such findings are not reported in all relevant studies, with a number of studies showing no difference between ADHD and TD children's sleep profiles (Bergwerff et al., 2016; Waldon et al., 2015; Wiebe et al., 2012). Such discrepancies may be explained by variations in actigraphic protocols applied (e.g., how long the actiwatch is worn for, is it worn during school days or free days, the analytic algorithms applied) as well as differences in study cohorts. Nonetheless, given the good tolerability of actigraphy and its suitability for application in the home setting, such approaches should be further encouraged in the future according to more standardized methodology (Smith et al., 2018). Further, most studies using actigraphy reviewed had sample sizes of 50 children or less in case control design, indicating a need for larger studies with sufficient statistical power to investigate issues such as the impacts of comorbid behavior disorders on sleep in children with ADHD as well as the impacts of environmental and family factors on sleep. Another important factor is the effect of the time of the year when the study has been conducted, (example Christmas (Langevin and Ramdé, 2012) or outside of school term-time) when the usual schedules of the child are disrupted due to social or familial events, or for specific days of the weekend or weekdays.

Some studies utilizing PSG report changes in sleep architecture in children with ADHD, including less time spent in REM, shorter REM sleep latency and lower frequency of eye movement during REM (Akinci et al., 2015; Díaz- Roman & Buela-Casal et al., 2019; Grissom et al., 2009; Gruber et al., 2009), although such findings are not ubiquitous (eg. Kirov et al., 2012, report greater REM duration in ADHD). For NREM features, lower periodic EEG activity (Cyclic Alternating Pattern, CAP) for stage 2 sleep was indicated along with higher 12 Hz frontal spindle power for ADHD children when compared to controls and its significant positive relationship with reaction time variability measuring attention (Akinci et al., 2015; De Dea et al., 2018; Grissom et al., 2009; Saito et al., 2019; Silvestri et al., 2009). Lower CAP rate was also found by Miano et al. (2006) in children with ADHD, perhaps indicating hyperarousal during NREM sleep in these children. Increased topographical distribution of Slow Wave Activity (SWA) localized over the central regions for ADHD children has been reported (Miano et al., 2019; Ringli et al., 2013) and previous studies have found that SWA has an age dependent shift that runs through the posterior-anterior axis between the age of 2 years and adolescence (Kurth et al., 2010; Novelli et al., 2016). Therefore, higher concentration of SWA in the central region among the ADHD children may reflect the neurodevelopmental nature of ADHD. However, findings of sleep EEG changes were not ubiquitous; Příhodová et al. (2012) reported no significant alteration in sleep REM or NREM parameters in ADHD. Small study cohort sizes and variations in study protocols (e.g., number of consecutive nights PSG recording, home of clinical research setting) may underpin such discrepancies. Another significant limitation for studies employing nocturnal polysomnography records is that they have done so for one night, thereby not controlling the first night effect, and studies which have used only sleep lab records and not for home environments might be subject to effects of a novel sleep environment on the sleep parameters measured (Galland et al., 2010; Miano et al., 2019; Silvestri et. al., 2009; Um et al., 2016). Further although studies have mentioned the washout periods for the child's ongoing ADHD medications, abstaining from the medication might influence the child's sleep which might be reflected in the records, and this significantly effects the total results, especially if the PSG is done for 1 night (Galland et al., 2010). It might prove beneficial to acknowledge the above lapses as EEG recordings during sleep have the potential to reveal important neurophysiological insights into ADHD and lead to understanding of how sleep changes may contribute to ADHD symptoms in children.

Our review of the current literature on circadian function in children with ADHD revealed a somewhat limited set of investigations, with significant variations in methodology and some inconsistent findings. For example, better ADHD clinical scores were associated with higher morning cortisol levels were indicated by some studies (Angeli et al., 2018; Wang et al., 2017) but not others (Imeraj et al., 2012; Buske-Kirschbaum et al., 2019). We uncovered only two investigations of melatonin rhythms in childhood ADHD, with inconsistent findings (Novokova et al., 2011; Paclt et al., 2011). Likewise, there was a low number of studies examining chronotype and/or diurnal preference in children with ADHD (Van der Heijden et al., 2018; Tarakçioğlu et al., 2018). For both of these studies subjective reports of chronotype were utilized which is not as accurate as the use of objective measures such as actigraphy, or assessing Dim Light Melatonin onset. Moreover, the studies in this section have used case-control design thereby limiting inference regarding long-term influence of sleep/ADHD associated behaviors on chronotype, which would be possible through longitudinal investigations. Our review found only 11 studies investigating circadian functions in ADHD children (10 case-control and 1 longitudinal). This lack of studies is striking in the context of the more developed literature in circadian function in adult ADHD (Coogan and McGowan, 2017), and may reflect the perceived difficulties of examining circadian rhythms in children. Given that the circadian system is a key shaper of sleep/wake behavior, this is an area that clearly warrants further research in ADHD.

Considering consequences of sleep problems in children with ADHD, the reviewed studies revealed consistent associations between anxiety/depression symptoms, as well as wider range internalizing and externalizing comorbidities, with sleep in children with ADHD (Becker et al., 2018; Blunden et al., 2011; Lucas et al., 2017; Lycett et al., 2016; Mulraney et al., 2016, 2017; Tong et al., 2018); such associations have also been previously indicated in adults with ADHD (Oğuztürk et al., 2013). There were also associations between poorer executive-cognitive functions and sleep deficits consistently reported in children with ADHD (Cremone, Kurdziel, et al., 2017; Hansen et al., 2013; Moreau et al., 2013). Cognitive functions assessed through manual/machine aided neuropsychological tests and sleep measures revealed cross sectional as well as longitudinal associations between these in ADHD (Cremone et al., 2018; Gruber et al., 2011; Kidwell et al., 2017; Saito et al., 2019; Sciberras et al., 2015; Surratt et al., 2011; Zambrano-sánchez et al., 2013). For example, Kidwell et al. (2017) found longitudinal associations between sleep problems at age 3 and higher levels of inattention/hyperactivity in 4th grade predicted by higher executive functions deficits. Further, sleep may be important for emotional regulation in ADHD. Cremone et al. (2018) hypothesized that positive attention biases may be associated with heightened reward sensitivity in children with ADHD, causing overreliance on (positive cues) rewards, which may contribute to behavioral manifestations for emotional dysregulation. These studies highlight the potential significance of impaired sleep for

cognition and emotional regulation in children with ADHD. However, the above findings should be interpreted with caution, as for most of the cross sectional studies (Lycett, Mensah, et al., 2015; Lycett et al., 2016; Lucas et al., 2019; Moreau et al., 2013; Sciberras et al., 2015; Thomas et al., 2018), the recruited ADHD children (with comorbid sleep concerns) might have presented higher level of ADHD symptom severity for which they were on medication, which in turn might have had an influence on the measured sleep feature. Additionally, the paucity of longitudinal studies exploring the developmental relationship between sleep functions and cognitive/emotional functions among ADHD children (our review located 6 such studies in this section) must be taken into consideration for future research.

Given that sleep disturbances appear to be consistently associated with cognitive and emotional problems in children with ADHD, it might be expected that cognitive/behavioral interventions that are designed to improve sleep may result in lessened ADHD symptoms. Behavioral sleep interventions improved caregiver rated sleep functions along with a proportion of sleep mediated improvements in ADHD symptoms, behavior and quality of life (Bériault et al., 2018; Hiscock et al., 2015; Papapdopoulos et al., 2019). However, we found that the evidence base for this area is underdeveloped (we found the above mentioned 3 studies exploring this field of intervention). Sleep trainings further brought about wide range of improvements in the child's sleep functions and related behaviors (Corkum et al., 2016; Keshavarzi et al., 2014; Peppers et al., 2016). Sleep hygiene interventions that have directly or simultaneously targeted sleep in ADHD had a beneficial effect on children's emotional, behavioral and social performance (Dewald-Kaufmann et al., 2013; Weiss et al., 2006). However, there is an absence of studies employing chronotherapeutic techniques for ADHD children that might involve using environmental stimuli to influence their biological clock. Also we noted only 6 randomized controlled trials, most of which had sample sizes in the range of 20 to 60 patients, again indicating a need for a more systematic approach involving larger (potentially multi-site) studies.

When examining the literature on the effects of ADHD pharmacotherapy on sleep, we found that a substantial number of studies (14 out of the 42 in this section) assessed sleep as a secondary outcome (often via a single item rating). 17 out of 42 of the included studies examined the effects of MPH, and a number of papers reported dose-dependent associations with greater sleep complaints, later bed times, negative effects on wake up time and total sleep time, day time sleepiness and higher insomnia (Becker, Pfiffner, et al., 2016; Childress et al., 2009; Cockcroft et al., 2009; Lee et al., 2011, 2012; Sonuga-Barke et al., 2009). However, some studies reported no negative effects of MPH on sleep (Ashkenasi 2011; Findling et al., 2009) and a few studies reported positive outcomes for sleep of MPH (Chin et al., 2018; Morash-Conway et al., 2017; Ricketts et al., 2018). Seven studies explored the effect of individual MPH preparations on sleep in childhood ADHD, with results not clearly indicating a preference for either extended or immediate release preparations (Kim 2010; Kim et al., 2011; Pelham et al., 2011; Pliszka et al., 2017). There were few studies assessing the sleep impacts of non-stimulant medication in children with ADHD (such as Atomoxetine, Guanfacine and Divalproex). The inconsistencies noted for MPH may be a function of the rudimentary characterization of sleep applied in many of these studies and indicates a need for more systematic assessment of MPH and other less commonly applied non-stimulant treatments on sleep in children.

Manifesto for Future Research in Sleep and childhood ADHD

Drawing together the points highlighted above, we present a manifesto for future research on sleep in children with ADHD. The first is that as sleep is a complex phenomenon with multiple phases that can be measured in multiple ways (each approach with important advantages and disadvantages) and fulfills multiple functions, the assessment of sleep in ADHD studies should be comprehensive and multifaceted. Such approaches should incorporate objective measures such as actigraphy and PSG in parallel with subjective measures such as parental report, and standardized protocols be applied when such consensus processes are available. Studies should be sufficiently well powered to allow for specified sub-group analysis (e.g., of ADHD subtypes). Secondly, proving causal relationships between sleep problems and functional outcomes in ADHD may not be achievable, rather a pragmatic approach of clinical utility may be adopted; that is to say the primary aim should be to develop and deploy effective interventions and further develop fundamental knowledge of the etiological and symptomatological profile of ADHD. There has been some interest recently in attempting to solve the "chicken and egg" question of sleep disturbance and ADHD; does one lead to the other, and if such in which order? (Bijlenga et al., 2019; Raman and Coogan, 2019). However, this may be a distinction that is not meaningful; for example, if sleep disturbance is part of the sytmptomatic profile of ADHD that manifests earlier than other symptoms during the developmental time course, then ascribing sleep changes as causal factors in the development of ADHD will not be justified. Future studies may examine the role of sleep parameters as state marker in ADHD during successful and unsuccessful treatment to hint at the relationship between sleep and the "classic" domains of inattention, hyperactivity and impulsivity. Further, we must consider whether there are generalized sleep disturbances associated with neuropsychiatric disorders, as whether there are specific features such as

delayed sleep timing or sleep maintenance problems that are more strongly associated with specific disorders such as ADHD (which has been argued to be the case in adult ADHD; Coogan and McGowan, 2017).

Thirdly, it should be borne in mind that mitigation of sleep problems, in and of themselves, may represent highly worthwhile goals, even if they are not accompanied by significant impacts on ADHD scores (e.g., by lessening sleep impacts in caregivers; Sacco et al., 2018). Fourthly, research should be directed to seemingly obvious questions relating to everyday clinical practice; an example of such an issue is whether time of dosing of different MPH formulations impacts sleep and ADHD symptoms differentially, as animal experiments indicate that this may be the case (Antle et al., 2012; Baird et al., 2013). Likewise, does time-of-day of testing on neuropsychological batteries impact on results in a clinically significant way, as is indicated in adults (Korman et al., 2019). Fifthly, when sleep behavioral interventions in ADHD are trialed they should be done so as blinded placebo controlled RCTs in multi-site studies to allow for sufficient statistical power; the literature as it is currently comprised is dominated by small, under-powered case-control studies that may provide tantalizing indications of promise and/or causal relationships, but are not useful in guiding clinical practice unless they are built on by larger studies. Lastly, majority of the studies in our review had lesser number of female participants as compared to males, thereby significantly limiting generalizability of findings to females. Keeping in mind the gender based variations in both macro and micro structure of sleep (Acebo et al., 1996; Sadeh et al., 2000), circadian rhythm markers (Boivin et al., 2016; Cain et al., 2010; Duffy et al., 2011; Santhi et al., 2016) and even the ADHD clinical pictures (Biederman et al., 2002), it is indeed indicative that the effect each variable has on the other might be based on such differences. Therefore, studies investigating sleep properties among ADHD children are recommended to have comparable number of female and male participants, where gender-based findings are discussed.

Conclusion

We have conducted the first comprehensive systematic review of sleep in children with ADHD, examining studies involving ~42,000 children. Our review highlights that reports of disturbed sleep in children with ADHD are common, that such disturbances may contribute to cognitive and emotional impairments and may be tractable through behavioral and cognitive sleep interventions. We have identified several important weaknesses and knowledge gaps in the literature and have laid out some points to be considered in the design and implementation of future studies of sleep in children with ADHD.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

ORCID iD

Andrew N. Coogan https://orcid.org/0000-0003-1998-0240

Supplemental Material

Supplemental material for this article is available online.

References

Abdelgadir, I. S., Gordon, M. A., & Akobeng, A. K. (2018). Melatonin for the management of sleep problems in children with neurodevelopmental disorders: A systematic review and metaanalysis. *Archives of Disease in Childhood*, *103*(12), 1155–1162.

Abou-Khadra, M. K., Amin, O. R., Shaker, O. G., & Rabah, T. M. (2013). Parent-reported sleep problems, symptom ratings, and serum ferritin levels in children with attention-deficit/hyperactivity disorder: A case control study. *BMC Pediatrics*, *13*(1), 217.

Acebo, C., Millman, R. P., Rosenberg, C., Cavallo, A., & Carskadon, M. A. (1996). Sleep, breathing, and cephalometrics in older children and young adults: Part I—Normative values. *Chest*, *109*(3), 664–672.

Ahmed, R., Borst, J., Wei, Y. C., & Aslani, P. (2017). Parents' perspectives about factors influencing adherence to pharmacotherapy for ADHD. *Journal of Attention Disorders*, *21*(2), 91–99.

Akinci, G., Oztura, I., Hiz, S., Akdogan, O., Karaarslan, D., Ozek, H., & Akay, A. (2015). Sleep structure in children with attention-deficit/hyperactivity disorder. *Journal of Child Neurology*, *30*(11), 1520–1525.

Alamar, H., Blackwell, J., Nash, H., Kellar, I., & Weighall, A. (2015). *A meta-analysis of behavioural interventions for sleep problems in children with neuro-developmental disorders. Conference presentation. Newcastle Sleep 2015. British Sleep Society*. Newcastle, United Kingdom.

Andersson, H., & Sonnesen, L. (2018). Sleepiness, occlusion, dental arch and palatal dimensions in children attention deficit hyperactivity disorder (ADHD). *European Archives of Paediatric Dentistry*, *19*(2), 91–97.

Angeli, E., Korpa, T., Johnson, E. O., Apostolakou, F., Papassotiriou, I., Chrousos, G. P., & Pervanidou, P. (2018). Salivary cortisol and alpha-amylase diurnal profiles and stress reactivity in children with attention deficit hyperactivity disorder. *Psychoneuroendocrinology*, *90*, 174–181.

Antle, M. C., Van Diepen, H. C., Deboer, T., Pedram, P., Pereira, R. R., & Meijer, J. H. (2012). Methylphenidate modifies the motion of the circadian clock. *Neuropsychopharmacology*, *37*(11), 2446–2455.

Aronen, E. T., Lampenius, T., Fontell, T., & Simola, P. (2014). Sleep in children with disruptive behavioral disorders. *Behavioral Sleep Medicine*, *12*(5), 373–388.

Ashkenasi, A. (2011). Effect of transdermal methylphenidate wear times on sleep in children with attention deficit hyperactivity disorder. *Pediatric Neurology*, *45*(6), 381–386.

Baird, A. L., Coogan, A. N., Kaufling, J., Barrot, M., & Thome, J. (2013). Daily methylphenidate and atomoxetine treatment impacts on clock gene protein expression in the mouse brain. *Brain Research*, *1513*, 61–71.

Ball, J. D., Tiernan, M., Janusz, J., & Furr, A. (1997). Sleep patterns among children with attentiondeficit hyperactivity disorder: A reexamination of parent perceptions. *Journal of Pediatric Psychology*, *22*(3), 389–398.

Becker, S. P., Froehlich, T. E., & Epstein, J. N. (2016). Effects of methylphenidate on sleep functioning in children with attention-deficit/hyperactivity disorder. *Journal of Developmental and Behavioral Pediatrics*, *37*(5), 395–404.

Becker, S. P., Pfiffner, L. J., Stein, M. A., Burns, G. L., & McBurnett, K. (2016). Sleep habits in children with attention-deficit/hyperactivity disorder predominantly inattentive type and associations with comorbid psychopathology symptoms. *Sleep Medicine*, *21*, 151–159.

Becker, S. P., Cusick, C. N., Sidol, C. A., Epstein, J. N., & Tamm, L. (2018). The impact of comorbid mental health symptoms and sex on sleep functioning in children with ADHD. *European Child & Adolescent Psychiatry*, *27*(3), 353–365.

Bergwerff, C. E., Luman, M., & Oosterlaan, J. (2016). No objectively measured sleep disturbances in children with attention deficit/hyperactivity disorder. *Journal of Sleep Research*, *25*(5), 534–540.

Bériault, M., Turgeon, L., Labrosse, M., Berthiaume, C., Verreault, M., Berthiaume, C., & Godbout, R. (2018). Comorbidity of ADHD and anxiety disorders in school-age children: Impact on sleep and response to a cognitive-behavioral treatment. *Journal of Attention Disorders*, *22*(5), 414–424.

Bessey, M., Coulombe, J. A., Smith, I. M., & Corkum, P. (2013). Assessing parental sleep attitudes and beliefs in typically developing children and children with ADHD and ASD. *Children's Health Care*, *42*(2), 116–133.

Bessey, M., Richards, J., & Corkum, P. (2013). Sleep lab adaptation in children with attention deficit/hyperactivity disorder and typically developing children. *Sleep Disorders*, *2013*, 698957.

Bestmann, A., Conzelmann, A., Baving, L., & Prehn-Kristensen, A. (2019). Associations between cognitive performance and sigma power during sleep in children with attention-deficit/hyperactivity disorder, healthy children, and healthy adults. *PLoS One*, *14*(10), e0224166.

Biederman, J., Mick, E., Faraone, S. V., Braaten, E., Doyle, A., Spencer, T., Wilens, T. E., Frazier, E., & Johnson, M. A. (2002). Influence of gender on attention deficit hyperactivity disorder in children referred to a psychiatric clinic. *American Journal of Psychiatry*, *159*(1), 36–42.

Bijlenga, D., Vollebregt, M. A., Kooij, J. S., & Arns, M. (2019). The role of the circadian system in the etiology and pathophysiology of ADHD: Time to redefine ADHD? *ADHD Attention Deficit and Hyperactivity Disorders*, *11*(1), 5–19.

Bioulac, S., Micoulaud-Franchi, J. A., & Philip, P. (2015). Excessive daytime sleepiness in patients with ADHD—Diagnostic and management strategies. *Current Psychiatry Reports*, *17*(8), 69.

Blader, J. C., Schooler, N. R., Jensen, P. S., Pliszka, S. R., & Kafantaris, V. (2009). Adjunctive divalproex versus placebo for children with ADHD and aggression refractory to stimulant monotherapy. *American Journal of Psychiatry*, *166*(12), 1392–1401.

Blumer, J. L., Findling, R. L., Shih, W. J., Soubrane, C., & Reed, M. D. (2009). Controlled clinical trial of zolpidem for the treatment of insomnia associated with attention-deficit/hyperactivity disorder in children 6 to 17 years of age. *Pediatrics*, *123*(5), e770–e776.

Blunden, S. L., Milte, C. M., & Sinn, N. (2011). Diet and sleep in children with attention deficit hyperactivity disorder: Preliminary data in Australian children. *Journal of Child Health Care*, *15*(1), 14–24.

Bock, D. E., Roach-Fox, E., Seabrook, J. A., Rieder, M. J., & Matsui, D. (2016). Sleep-promoting medications in children: Physician prescribing habits in Southwestern Ontario, Canada. *Sleep Medicine*, *17*, 52–56.

Boivin, D. B., Shechter, A., Boudreau, P., Begum, E. A., & Ying-Kin, N. M. K. N. (2016). Diurnal and circadian variation of sleep and alertness in men vs. naturally cycling women. *Proceedings of the National Academy of Sciences*, *113*(39), 10980–10985.

Borbély, A. A., Daan, S., Wirz-Justice, A., & Deboer, T. (2016). The two-process model of sleep regulation: A reappraisal. *Journal of Sleep Research*, *25*(2), 131–143.

Buchhorn, R., Conzelmann, A., Willaschek, C., Störk, D., Taurines, R., & Renner, T. J. (2012). Heart rate variability and methylphenidate in children with ADHD. *ADHD Attention Deficit and Hyperactivity Disorders*, *4*(2), 85–91.

Buske-Kirschbaum, A., Trikojat, K., Tesch, F., Schmitt, J., Roessner, V., Luksch, H., Rösen-Wolff, A., & Plessow, F. (2019). Altered hypothalamus-pituitary-adrenal axis function: A relevant factor in the comorbidity of atopic eczema and attention deficit/hyperactivity disorder? *Psychoneuroendocrinology*, *105*, 178–186.

Cain, S. W., Dennison, C. F., Zeitzer, J. M., Guzik, A. M., Khalsa, S. B. S., Santhi, N., . . . Duffy, J. F. (2010). Sex differences in phase angle of entrainment and melatonin amplitude in humans. *Journal of Biological Rhythms*, *25*(4), 288–296.

Chiang, H. L., Gau, S. S. F., Ni, H. C., Chiu, Y. N., Shang, C. Y., Wu, Y. Y., Lin, L. Y., Tai, Y. M., & Soong, W. T. (2010). Association between symptoms and subtypes of attention-deficit hyperactivity disorder and sleep problems/disorders. *Journal of Sleep Research*, *19*(4), 535–545.

Childress, A. C., Spencer, T., Lopez, F., Gerstner, O., Thulasiraman, A., Muniz, R., & Post, A. (2009). Efficacy and safety of dexmethylphenidate extended-release capsules administered once daily to children with attention-deficit/hyperactivity disorder. *Journal of Child and Adolescent Psychopharmacology*, *19*(4), 351–361.

Chin, W. C., Huang, Y. S., Chou, Y. H., Wang, C. H., Chen, K. T., Hsu, J. F., & Hsu, S. C. (2018). Subjective and objective assessments of sleep problems in children with attention

deficit/hyperactivity disorder and the effects of methylphenidate treatment. *Biomedical Journal*, *41*(6), 356–363.

Chiraphadhanakul, K., Jaimchariyatam, N., Pruksananonda, C., & Chonchaiya, W. (2015). Increased sleep disturbances in Thai children with attention-deficit hyperactivity disorder compared with typically developing children. *Behavioral Sleep Medicine*, *14*, 677–686.

Choi, J., Yoon, I. Y., Kim, H. W., Chung, S., & Yoo, H. J. (2010). Differences between objective and subjective sleep measures in children with attention deficit hyperactivity disorder. *Journal of Clinical Sleep Medicine*, *6*(6), *589*−595.

Cockcroft, K., Ashwal, J., & Bentley, A. (2009). Sleep and daytime sleepiness in methylphenidate medicated and un-medicated children with attention-deficit/hyperactivity disorder (ADHD). *African Journal of Psychiatry*, *12*, 275–279.

Connor, D. F., Findling, R. L., Kollins, S. H., Sallee, F., López, F. A., Lyne, A., & Tremblay, G. (2010). Effects of guanfacine extended release on oppositional symptoms in children aged 6–12 years with attention-deficit hyperactivity disorder and oppositional symptoms. *CNS Drugs*, *24*(9), *755*−768.

Coogan, A. N., & McGowan, N. M. (2017). A systematic review of circadian function, chronotype and chronotherapy in attention deficit hyperactivity disorder. *ADHD Attention Deficit and Hyperactivity Disorders*, *9*(3), 129–147.

Corkum, P., Lingley-Pottie, P., Davidson, F., McGrath, P., Chambers, C. T., Mullane, J., Laredo, S., Woodford, K., & Weiss, S. K. (2016). Better nights/better days—Distance intervention for insomnia in school-aged children with/without ADHD: A randomized controlled trial. *Journal of Pediatric Psychology*, *41*(6), 701–713.

Corkum, P., Tannock, R., & Moldofsky, H. (1998). Sleep disturbances in children with attention-deficit/hyperactivity disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*, *37*(6), 637–646.

Cortese, S., Adamo, N., Giovane, C. D., Mohr-Jensen, C., Hayes, A. J., Carucci, S., Atkinson, L. Z., Tessari, L., Banaschewski, T., Coghill, D., Hollis, C., Simonoff, E., Zuddas, A., Barbui, C., Purgato, M., Steinhausen, H. C., Shokraneh, F., Xia, J., & Cipriani, A. (2018). Comparative efficacy and tolerability of medications for attention-deficit hyperactivity disorder in children, adolescents, and adults: A systematic review and network meta-analysis. *The Lancet Psychiatry*, *5*(9), 727−738.

Cortese, S., Brown, T. E., Corkum, P., Gruber, R., O'Brien, L. M., Stein, M., Weiss, M., & Owens, J. (2013). Assessment and management of sleep problems in youths with attention deficit/hyperactivity disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, *52*, 784–796.

Cortese, S., Konofal, E., Yateman, N., Mouren, M., & Lecendreux, M. (2006). Sleep and alertness in children with attention-deficit/hyperactivity disorder: A systematic review of the literature. *Sleep*, *29*(4), 504.

Cremone, A., Kurdziel, L. B., Fraticelli-Torres, A., McDermott, J. M., & Spencer, R. M. (2017). Napping reduces emotional attention bias during early childhood. *Developmental Science*, *20*(4), e12411.

Cremone, A., Lugo-Candelas, C. I., Harvey, E. A., McDermott, J. M., & Spencer, R. M. (2017). REM theta activity enhances inhibitory control in typically developing children but not children with ADHD symptoms. *Experimental Brain Research*, *235*(5), 1491–1500.

Cremone, A., Lugo-Candelas, C. I., Harvey, E. A., McDermott, J. M., & Spencer, R. M. (2018). Positive emotional attention bias in young children with symptoms of ADHD. *Child Neuropsychology*, *24*(8), 1137–1145.

De Dea, F., Zanus, C., Carrozzi, M., Stecca, M., & Accardo, A. (2018, July). *Characteristics of EEG power spectrum during sleep spindle events in ADHD children*. In *2018 40th annual international conference of the IEEE engineering in medicine and biology society (EMBC)* (pp. 1456–1459). IEEE.

Deboer, T. (2018). Sleep homeostasis and the circadian clock: Do the circadian pacemaker and the sleep homeostat influence each other's functioning? *Neurobiology of Sleep and Circadian Rhythms*, *5*, 68–77. https://doi.org/10.1016/j.nbscr.2018.02.003

Dewald-Kaufmann, J. F., Oort, F. J., & Meijer, A. M. (2013). The effects of sleep extension on sleep and cognitive performance in adolescents with chronic sleep reduction: An experimental study. *Sleep Medicine*, *14*(6), 510–517.

Díaz-Román, A., Mitchell, R., & Cortese, S. (2018). Sleep in adults with ADHD: Systematic review and meta-analysis of subjective and objective studies. *Neuroscience & Biobehavioral Reviews*, *89*, 61–71.

Díaz-Román, A., & Buela-Casal, G. (2019). Shorter REM latency in children with attention-deficit/hyperactivity disorder. *Psychiatry Research*, *278*, 188-193.

Duffy, J. F., Cain, S. W., Chang, A. M., Phillips, A. J., Münch, M. Y., Gronfier, C., . . . Czeisler, C. A. (2011). Sex difference in the near-24-hour intrinsic period of the human circadian timing system. *Proceedings of the National Academy of Sciences*, *108*(Supplement 3), 15602–15608.

Efron, D., Lycett, K., & Sciberras, E. (2014). Use of sleep medication in children with ADHD. *Sleep Medicine*, *15*(4), 472–475.

England, S. J., Picchietti, D. L., Couvadelli, B. V., Fisher, B. C., Siddiqui, F., Wagner, M. L., Hening, W. A., Lewin, D., Winnie, G., Cohen, B., & Walters, A. S. (2011). L-dopa improves restless legs syndrome and periodic limb movements in sleep but not attention-deficit-hyperactivity disorder in a double-blind trial in children. *Sleep Medicine*, *12*(5), 471–477.

Eyuboglu, M., & Eyuboglu, D. (2018). Behavioral sleep problems in previously untreated children with attention deficit hyperactivity disorder. *Psychiatry and Clinical Psychopharmacology*, *28*(1), 19–24.

Faraone, S. V., Glatt, S. J., Bukstein, O. G., Lopez, F. A., Arnold, L. E., & Findling, R. L. (2009). Effects of once-daily oral and transdermal methylphenidate on sleep behavior of children with ADHD. *Journal of Attention Disorders*, *12*(4), 308–315.

Ferri, R., Bruni, O., Novelli, L., Picchietti, M. A., & Picchietti, D. L. (2013). Time structure of leg movement activity during sleep in attention-deficit/hyperactivity disorder and effects of levodopa. *Sleep Medicine*, *14*(4), 359–366.

Findling, R. L., Wigal, S. B., Bukstein, O. G., Boellner, S. W., Abikoff, H. B., Turnbow, J. M., & Civil, R. (2009). Long-term tolerability of the methylphenidate transdermal system in pediatric attention-deficit/hyperactivity disorder: A multicenter, prospective, 12-month, open-label, uncontrolled, phase III extension of four clinical trials. *Clinical Therapeutics*, *31*(8), 1844–1855.

Galán, C., Sánchez, S., Franco, L., Bravo, R., Rivero, M., Rodríguez, A. B., & Barriga, C. (2017). Tryptophan-enriched antioxidant cereals improve sleep in children with autistic spectrum and attention deficit hyperactivity disorders. *Journal of Cellular Neuroscience & Oxidative Stress*, *9*(1), 608–616.

Galland, B. C., Tripp, E. G., & Taylor, B. J. (2010). The sleep of children with attention deficit hyperactivity disorder on and off methylphenidate: A matched case-control study. *Journal of Sleep Research*, *19*, 366–373.

Galland, B. C., Tripp, E. G., Gray, A., & Taylor, B. J. (2011). Apnea-hypopnea indices and snoring in children diagnosed with ADHD: a matched case-control study. *Sleep and Breathing*, *15*(3), 455-462.

Geladé, K., Janssen, T. W., Bink, M., Twisk, J. W., van Mourik, R., Maras, A., & Oosterlaan, J. (2018). A 6-month follow-up of an RCT on behavioral and neurocognitive effects of neurofeedback in children with ADHD. *European Child & Adolescent Psychiatry*, *27*(5), 581–593.

Giblin, J. M., & Strobel, A. L. (2011). Effect of lisdexamfetamine dimesylate on sleep in children with ADHD. *Journal of Attention Disorders*, *15*(6), 491–498.

Gomes, A. A., Parchão, C., Almeida, A., Clemente, V., & De Azevedo, M. H. P. (2014). Sleep–wake patterns reported by parents in hyperactive children diagnosed according to ICD-10, as compared to paired controls. *Child Psychiatry & Human Development*, *45*(5), 533–543.

Gregory, A. M., Agnew-Blais, J. C., Matthews, T., Moffitt, T. E., & Arseneault, L. (2017). ADHD and sleep quality: Longitudinal analyses from childhood to early adulthood in a twin cohort. *Journal of Clinical Child & Adolescent Psychology*, *46*(2), 284–294.

Grissom, E. M., Brubaker, B., Capdevila, O. S., Hawley, W. R., & Gozal, D. (2009). Eye movement during REM sleep in children with attention deficit hyperactivity disorder. *Developmental Neuropsychology*, *34*(5), 552–559.

Gruber, R., Fontil, L., Bergmame, L., Wiebe, S. T., Amsel, R., Frenette, S., & Carrier, J. (2012). Contributions of circadian tendencies and behavioral problems to sleep onset problems of children with ADHD. *BMC Psychiatry*, *12*(1), 212.

Gruber, R., Michaelsen, S., Bergmame, L., Frenette, S., Bruni, O., Fontil, L., & Carrier, J. (2012). Short sleep duration is associated with teacher-reported inattention and cognitive problems in healthy school-aged children. *Nature Science of Sleep*, *4*, 33–40.

Gruber, R., Wiebe, S., Montecalvo, L., Brunetti, B., Amsel, R., & Carrier, J. (2011). Impact of sleep restriction on neurobehavioral functioning of children with attention deficit hyperactivity disorder. *Sleep*, *34*(3), 315–323.

Gruber, R., Xi, T., Frenette, S., Robert, M., Vannasinh, P., & Carrier, J. (2009). Sleep disturbances in prepubertal children with attention deficit hyperactivity disorder: A home polysomnography study. *Sleep*, *32*(3), 343–350.

Grünwald, J., & Schlarb, A. A. (2017). Relationship between subtypes and symptoms of ADHD, insomnia, and nightmares in connection with quality of life in children. *Neuropsychiatric Disease and Treatment*, *13*, 2341.

Hansen, B. H., Skirbekk, B., Oerbeck, B., Richter, J., & Kristensen, H. (2011). Comparison of sleep problems in children with anxiety and attention deficit/hyperactivity disorders. *European Child & Adolescent Psychiatry*, *20*(6), 321.

Hansen, B. H., Skirbekk, B., Oerbeck, B., Wentzel-Larsen, T., & Kristensen, H. (2013). Persistence of sleep problems in children with anxiety and attention deficit hyperactivity disorders. *Child Psychiatry & Human Development*, *44*(2), 290–304.

Hegarty, C. E., Gupta, M. W., Miller, E., Terashima, K., Loo, S., McCracken, J., McGough, J., Bilder, R. M., Poldrack, R. A., Galvan, A., & Bookheimer, S. (2019). Symptom severity in youths with attention deficit hyperactivity disorder associated with normalizing effects of treatment on fMRI response during a stop signal task. Advance online publication. https://doi.org/10.1101/599803.

Henriques Filho, P. S. A. (2016). Sleep disorder investigation might be considered to be mandatory in attention deficit/hyperactivity disorder guideline. *Arquivos de Neuro-Psiquiatria*, *74*(9), 701–707.

Hiscock, H., Sciberras, E., Mensah, F., Gerner, B., Efron, D., Khano, S., & Oberklaid, F. (2015). Impact of a behavioral sleep intervention on symptoms and sleep in children with attention deficit hyperactivity disorder, and parental mental health: Randomised controlled trial. *BMJ*, *350*, h68.

Hoebert, M., Van Der Heijden, K. B., Van Geijlswijk, I. M., & Smits, M. G. (2009). Long-term follow-up of melatonin treatment in children with ADHD and chronic sleep onset insomnia. *Journal of Pineal Research*, *47*(1), 1–7.

Hvolby, A. (2015). Associations of sleep disturbance with ADHD: implications for treatment. *ADHD Attention Deficit and Hyperactivity Disorders*, *7*(1), 1-18.

Hvolby, A., & Bilenberg, N. (2011). Use of ball blanket in attention-deficit/hyperactivity disorder sleeping problems. *Nordic Journal of Psychiatry*, *65*(2), 89–94.

Hvolby, A., Jorgensen, J., & Bilenberg, N. (2009). Parental rating of sleep in children with attention deficit/hyperactivity disorder. *European Child Adolescent Psychiatry*, *18*, 429–438.

Imeraj, L., Antrop, I., Roeyers, H., Deschepper, E., Bal, S., & Deboutte, D. (2011). Diurnal variations in arousal: A naturalistic heart rate study in children with ADHD. *European Child & Adolescent Psychiatry*, *20*(8), 381–392.

Imeraj, L., Antrop, I., Roeyers, H., Swanson, J., Deschepper, E., Bal, S., & Deboutte, D. (2012). Time-of-day effects in arousal: Disrupted diurnal cortisol profiles in children with ADHD. *Journal of Child Psychology and Psychiatry*, *53*(7), 782–789.

Ironside, S., Davidson, F., & Corkum, P. (2010). Circadian motor activity affected by stimulant medication in children with attention-deficit/hyperactivity disorder. *Journal of Sleep Research*, *19*(4), 546–551.

Jackson, D. B., & Vaughn, M. G. (2017). Sleep and preteen delinquency: Is the association robust to ADHD symptomatology and ADHD diagnosis? *Journal of Psychopathology and Behavioral Assessment*, *39*(4), 585–595.

Keshavarzi, Z., Bajoghli, H., Mohamadi, M. R., Salmanian, M., Kirov, R., Gerber, M., Holsboer-Trachsler, E., & Brand, S. (2014). In a randomized case-control trial with 10-years olds suffering from attention deficit/hyperactivity disorder (ADHD) sleep and psychological functioning improved

during a 12-week sleep-training program. *World Journal of Biological Psychiatry*, *15*, 609–619.

Kidwell, K. M., Hankey, M., Nelson, J. M., Espy, K. A., & Nelson, T. D. (2017). Child executive control as a moderator of the longitudinal association between sleep problems and subsequent attention-deficit/hyperactivity disorder symptoms. *Journal of Pediatric Psychology*, *42*(10), 1144–1155.

Kim, W. H. (2010). The effect of OROS methylphenidate on the sleep of children with attentiondeficit/hyperactivity disorder. *International Clinical Psychopharmacology*, *25*(2), 107–115.

Kim, B. N., Kim, Y. N., Cheong, U. S., Kim, J. W., Hwang, J. W., Shin, M. S., & Cho, S. C.; Korean OROSMPH Study Group. (2011). Switching from methylphenidate-immediate release (MPH-IR) to methylphenidate-OROS (OROS-MPH): A multi-center, open-label study in Korea. *Clinical Psychopharmacology and Neuroscience*, *9*(1), 29.

Kirov, R., Uebel, H., Albrecht, B., Banaschewski, T., Yordanova, J., & Rothenberger, A. (2012). Attention-deficit/hyperactivity disorder (ADHD) and adaptation night as determinants of sleep patterns in children. *European Child & Adolescent Psychiatry*, *21*(12), 681–690.

Knight, F. L. C., & Dimitriou, D. (2019). Poor sleep has negative implications for children with and without ADHD, but in different ways. *Behavioral Sleep Medicine*, *17*(4), 423–436.

Konofal, E., Lecendreux, M., Bouvard, M. P., & Mouren-Simeoni, M. C. (2001). High levels of nocturnal activity in children with attention-deficit hyperactivity disorder: A video analysis. *Psychiatry and Clinical Neurosciences*, *55*(2), 97–103.

Konofal, E., Lecendreux, M., & Cortese, S. (2010). Sleep and ADHD. *Sleep Medicine*, *11*(7), 652–658.

Korman, M., Levy, I., Maaravi-Hesseg, R., Eshed-Mantel, A., & Karni, A. (2019). Subclinical scores in self-report based screening tools for attention deficits correlate with cognitive traits in typical evening-type adults tested in the morning. *Frontiers in Psychology*, *10*, 1397.

Krause, A. J., Simon, E. B., Mander, B. A., Greer, S. M., Saletin, J. M., Goldstein-Piekarski, A. N., & Walker, M. P. (2017). The sleep-deprived human brain. *Nature Reviews Neuroscience*, *18*(7), 404–418.

Krinzinger, H., Hall, C. L., Groom, M. J., Ansari, M. T., Banaschewski, T., Buitelaar, J. K., . . . Falissard, B. (2019). Neurological and psychiatric adverse effects of long-term methylphenidate treatment in ADHD: A map of the current evidence. *Neuroscience & Biobehavioral Reviews*, *107*, 945–968.

Kurth, S., Ringli, M., Geiger, A., LeBourgeois, M., Jenni, O. G., & Huber, R. (2010). Mapping of cortical activity in the first two decades of life: A high-density sleep electroencephalogram study. *Journal of Neuroscience*, *30*(40), 13211–13219.

Kwon, S., Sohn, Y., Jeong, S. H., Chung, U. S., & Seo, H. (2014). Prevalence of restless legs syndrome and sleep problems in Korean children and adolescents with attention deficit hyperactivity disorder: a single institution study. *Korean Journal of Pediatrics*, *57*(7), 317.

Langevin, R., & Ramdé, J. (2012). Attention deficit hyperactivity disorder (ADHD) in children, seasonal photoperiods, nocturnal movements and diurnal agitation. *Journal of the Canadian Academy of Child and Adolescent Psychiatry*, *21*(1), 53–58.

Lee, J., Grizenko, N., Bhat, V., Sengupta, S., Polotskaia, A., & Joober, R. (2011). Relation between therapeutic response and side effects induced by methylphenidate as observed by parents and teachers of children with ADHD. *BMC Psychiatry*, *11*(1), 70.

Lee, H. K., Jeong, J. H., Kim, N. Y., Park, M. H., Kim, T. W., Seo, H. J., Lim, H. K., Hong, S. C., & Han, J. H. (2014). Sleep and cognitive problems in patients with attention-deficit hyperactivity disorder. *Neuropsychiatric Disease and Treatment*, *10*, 1799–1805.

Lee, S. H., Seo, W. S., Sung, H. M., Choi, T. Y., Kim, S. Y., Choi, S. J., Koo, B. H., & Lee, J. H. (2012). Effect of methylphenidate on sleep parameters in children with ADHD. *Psychiatry Investigation*, *9*(4), 384–390.

Liberati, A., Altman, D. G., Tetzlaff, J., Mulrow, C., Gøtzsche, P. C., Ioannidis, J. P., Clarke, M., Devereaux, P. J., Kleijnen, J., & Moher, D. (2009). The PRISMA statement for reporting systematic reviews and meta-analyses of studies that evaluate health care interventions: Explanation and elaboration. *Journal of Clinical Epidemiology*, *62*(10), e1–e34.

Lucas, I., Mulraney, M., & Sciberras, E. (2019). Sleep problems and daytime sleepiness in children with ADHD: Associations with social, emotional, and behavioral functioning at school, a crosssectional study. *Behavioral Sleep Medicine*, *17*(4), 411–422.

Lugo, J., Fadeuilhe, C., Gisbert, L., Setien, I., Delgado, M., Corrales, M., Richarte, V., & Ramos-Quiroga, J. A. (2020). Sleep in adults with autism spectrum disorder and attention deficit/hyperactivity disorder: A systematic review and meta-analysis. *European Neuropsychopharmacology*, *38*, 1–24.

Lycett, K., Mensah, F. K., Hiscock, H., & Sciberras, E. (2014). A prospective study of sleep problems in children with ADHD. *Sleep Medicine*, *15*(11), 1354–1361.

Lycett, K., Mensah, F. K., Hiscock, H., & Sciberras, E. (2015). Comparing subjective measures of behavioral sleep problems in children with ADHD: A cross-sectional study. *Sleep Medicine*, *16* (11), 1377–1380.

Lycett, K., Sciberras, E., Hiscock, H., & Mensah, F. K. (2016). Sleep problem trajectories and wellbeing in children with attention-deficit hyperactivity disorder: A prospective cohort study. *Journal of Developmental & Behavioral Pediatrics*, *37*(5), 405–414.

Lycett, K., Sciberras, E., Mensah, F. K., Gulenc, A., & Hiscock, H. (2014). Behavioral sleep problems in children with attention-deficit/hyperactivity disorder (ADHD): Protocol for a prospective cohort study. *BMJ Open*, *4*(2), e004070.

Lycett, K., Sciberras, E., Mensah, F. K., & Hiscock, H. (2015). Behavioral sleep problems and internalizing and externalizing comorbidities in children with attention-deficit/hyperactivity disorder. *European Child & Adolescent Psychiatry*, *24*(1), 31–40.

Lyon, M. R., Kapoor, M. P., & Juneja, L. R. (2011). The effects of L-theanine (Suntheanine®) on objective sleep quality in boys with attention deficit hyperactivity disorder (ADHD): A randomized, double-blind, placebo-controlled clinical trial. *Alternative Medicine Review*, *16*(4), 348.

Markovska-Simoska, S., & Pop-Jordanova, N. (2017). Quantitative EEG in children and adults with attention deficit hyperactivity

disorder: Comparison of absolute and relative power spectra and theta/beta ratio. *Clinical EEG and Neuroscience*, *48*(1), 20–32.

Martins, R., Scalco, J. C., Junior, G. J. F., da Silva Gerente, J. G., da Lapa Costa, M., & Beltrame, T. S. (2019). Sleep disturbance in children with attention-deficit hyperactivity disorder: A systematic review. *Sleep Science*, *12*(4), 295.

Martin, C. A., Papadopoulos, N., Chellew, T., Rinehart, N. J., & Sciberras, E. (2019). Associations between parenting stress, parent mental health and child sleep problems for children with ADHD and ASD: Systematic review. *Research in Developmental Disabilities*, *93*, 103463.

Masi, G., Fantozzi, P., Villafranca, A., Tacchi, A., Ricci, F., Ruglioni, L., Inguaggiato, E., Pfanner, C., & Cortese, S. (2019). Effects of melatonin in children with attention-deficit/hyperactivity disorder with sleep disorders after methylphenidate treatment. *Neuropsychiatric Disease and Treatment*, *15*, 663.

Matsuoka, M., Nagamitsu, S., Iwasaki, M., Iemura, A., Yamashita, Y., Maeda, M., Kitani, S., Kakuma, T., Uchimura, N., & Matsuishi, T. (2014). High incidence of sleep problems in children with developmental disorders: Results of a questionnaire survey in a Japanese elementary school. *Brain and Development*, *36*(1), 35–44.

Mayes, S. D., Calhoun, S. L., Bixler, E. O., Vgontzas, A. N., Mahr, F., Hillwig-Garcia, J., Elamir, B., Edhere-Ekezie, L., & Parvin, M. (2009). ADHD subtypes and comorbid anxiety, depression, and oppositional-defiant disorder: Differences in sleep problems. *Journal of Pediatric Psychology*, *34*(3), 328–337.

McGough, J. (2014). ADHD: ADHD. https://ebookcentral.proquest.com

Miano, S., Amato, N., Foderaro, G., Pezzoli, V., Ramelli, G. P., Toffolet, L., & Manconi, M. (2019). Sleep phenotypes in attention deficit hyperactivity disorder. *Sleep Medicine*, *60*, 123–131.

Miano, S., Donfrancesco, R., Bruni, O., Ferri, R., Galiffa, S., Pagani, J., Montemitro, E., Kheirandish, L., Gozal, D., & Villa, M. P. (2006). NREM sleep instability is reduced in children with attention-deficit/hyperactivity disorder. *Sleep*, *29*(6), 797–803.

Miano, S., Esposito, M., Foderaro, G., Ramelli, G. P., Pezzoli, V., & Manconi, M. (2016). Sleep related disorders in children with attention-deficit hyperactivity disorder: Preliminary results of a full sleep assessment study. *CNS Neuroscience & Therapeutics*, *22*(11), 906–914.

Mogavero, F., Jager, A., & Glennon, J. C. (2018). Clock genes, ADHD and aggression. *Neuroscience & Biobehavioral Reviews*, *91*, 51–68.

Mohammadpour, N., Jazayeri, S., Tehrani-Doost, M., Djalali, M., Hosseini, M., Effatpanah, M., Davari-Ashtiani, R, & Karami, E. (2018). Effect of vitamin D supplementation as adjunctive therapy to methylphenidate on ADHD symptoms: A randomized, double blind, placebo-controlled trial. *Nutritional Neuroscience*, *21*(3), 202–209.

Mohammadzadeh, S., Baghi, N., Yousefi, F., & Yousefzamani, B. (2019). Effect of omega-3 plus methylphenidate as an alternative therapy to reduce attention deficit-hyperactivity disorder in children. *Korean Journal of Pediatrics*, *62*(9), 360.

Montoya, A., Hervas, A., Cardo, E., Artigas, J., Mardomingo, M. J., Alda, J. A., Gastaminza, X., García-Polavieja, M. J., Gilaberte, I., & Escobar, R. (2009). Evaluation of atomoxetine for first-line treatment of newly diagnosed, treatment-naïve children and adolescents with attention deficit/hyperactivity disorder. *Current Medical Research and Opinion*, *25*(11), 2745–2754.

Morash-Conway, J., Gendron, M., & Corkum, P. (2017). The role of sleep quality and quantity in moderating the effectiveness of medication in the treatment of children with ADHD. *ADHD Attention Deficit and Hyperactivity Disorders*, *9*(1), 31–38.

Moreau, V., Rouleau, N., & Morin, C. M. (2013). Sleep, attention, and executive functioning in children with attention-deficit/hyperactivity disorder. *Archives of Clinical Neuropsychology*, *28*, 692–699.

Moreau, V., Rouleau, N., & Morin, C. M. (2014). Sleep of children with attention deficit hyperactivity disorder: Actigraphic and parental reports. *Behavioral Sleep Medicine*, *12*(1), 69–83.

Mulraney, M., Giallo, R., Lycett, K., Mensah, F., & Sciberras, E. (2016). The bidirectional relationship between sleep problems and internalizing and externalizing problems in children with ADHD: A prospective cohort study. *Sleep Medicine*, *17*, 45–51.

Mulraney, M., Zendarski, N., Mensah, F., Hiscock, H., & Sciberras, E. (2017). Do early internalizing and externalizing problems predict later irritability in adolescents with attention-deficit/hyperactivity disorder? *Australian & New Zealand Journal of Psychiatry*, *51*(4), 393–402.

Newcorn, J. H., Stein, M. A., Childress, A. C., Youcha, S., White, C., Enright, G., & Rubin, J. (2013). Randomized, double-blind trial of guanfacine extended release in children with attention-deficit/hyperactivity disorder: morning or evening administration. *Journal of the American Academy of Child & Adolescent Psychiatry*, *52*(9), 921-930.

Nikles, J., Mitchell, G. K., de Miranda Araújo, R., Harris, T., Heussler, H. S., Punja, S., . . . Senior, H. E. J. (2020). A systematic review of the effectiveness of sleep hygiene in children with ADHD. *Psychology, Health & Medicine*, *25*(4), 497-518.

Noble, G. S., O'Laughlin, L., & Brubaker, B. (2011). Attention deficit hyperactivity disorder and sleep disturbances: Consideration of parental influence. *Behavioral Sleep Medicine*, *10*, 41–53.

Novakova, M., Paclt, I., Ptacek, R., Kuzelova, H., Hajek, I., & Sumova, A. (2011). Salivary melatonin rhythm as a marker of the circadian system in healthy children and those with attention deficit/hyperactivity disorder. *Chronobiology International*, *28*, 630–637.

Novelli, L., D'atri, A., Marzano, C., Finotti, E., Ferrara, M., Bruni, O., & De Gennaro, L. (2016). Mapping changes in cortical activity during sleep in the first 4 years of life. *Journal of Sleep Research*, *25*(4), 381–389.

O'Callaghan, F. V., Al Mamun, A., O'Callaghan, M., Clavarino, A., Williams, G. M., Bor, W., Heussler, H., & Najman, J. M. (2010). The link between sleep problems in infancy and early childhood and attention problems at 5 and 14 years: Evidence from a birth cohort study. *Early Human Development*, *86*(7), 419–424.

Oğuztürk, Ö., Ekici, M., Çimen, D., Ekici, A., & Senturk, E. (2013). Attention deficit/hyperactivity disorder in adults with sleep apnea. *Journal of Clinical Psychology in Medical Settings*, *20*(2), 234–239.

Paclt, I., Ptacek, R., Kuzelova, H., Cermáková, N., Trefilová, A., Kollárová, P., & Cihal, L. (2011). Circadian rhythms of saliva melatonin in ADHD, anxious and normal children. *Neuroendocrinology Letters*, *32*(6), 790.

Papadopoulos, N., Sciberras, E., Hiscock, H., Mulraney, M., McGillivray, J., & Rinehart, N. (2019). The efficacy of a brief behavioral sleep intervention in school-aged children with ADHD and comorbid autism spectrum disorder. *Journal of Attention Disorders*, *23*(4), 341–350.

Pastor, P. N., & Reuben, C. A. (2008). *Diagnosed attention deficit hyperactivity disorder and learning disability: United States, 2004-2006. Data from the national health interview survey. Vital and health statistics* (Vol. 237(10)). Centers for Disease Control and Prevention.

Pelham, W. E., Waxmonsky, J. G., Schentag, J., Ballow, C. H., Panahon, C. J., Gnagy, E. M., Hoffman, M. T., Burrows-MacLean, L., Meichenbaum, D. L., Forehand, G. L., Fabiano, G. A., Tresco, K. E., Lopez-Williams, A., Coles, E. K., & González, M. A. (2011). Efficacy of a methylphenidate transdermal system versus tid methylphenidate in a laboratory setting. *Journal of Attention Disorders*, *15*(1), 28–35.

Pelsser, L. M., Frankena, K., Buitelaar, J. K., & Rommelse, N. N. (2010). Effects of food on physical and sleep complaints in children with ADHD: A randomised controlled pilot study. *European Journal of Pediatrics*, *169*(9), 1129–1138.

Peppers, K. H., Eisbach, S., Atkins, S., Poole, J. M., & Derouin, A. (2016). An intervention to promote sleep and reduce ADHD symptoms. *Journal of Pediatric Health Care*, *30*(6), e43–e48.

Peralta, G. P. (2018). Sleeping, TV, cognitively stimulating activities, physical activity, and attention-deficit hyperactivity disorder symptom incidence in children: A prospective study. *Journal of Developmental Behavioral Paediatrics*, *39*(3), 192–199.

Pliszka, S. R., Wilens, T. E., Bostrom, S., Arnold, V. K., Marraffino, A., Cutler, A. J., López, F. A., DeSousa, N. J., Sallee, F. R., Incledon, B., & Newcorn, J. H. (2017). Efficacy and safety of HLD200, delayed-release and extended-release methylphenidate, in children with attention-deficit/hyperactivity disorder. *Journal of Child and Adolescent Psychopharmacology*, *27*(6), 474–482.

Polmann, H., Réus, J. C., Maia, I., Dick, B., Gozal, D., Flores-Mir, C., . . . Canto, G. D. L. (2020). Association between sleep disordered breathing and symptoms of attention deficits in adults: a systematic review. *Sleep Medicine*. *73*, 223–230.

Poirier, A., & Corkum, P. (2018). Night-to-night variability of sleep in children with ADHD and typically developing controls. *Journal of Attention Disorders*, *22*(10), 942–946.

Pokhrel, P., Herzog, T. A., Black, D. S., Zaman, A., Riggs, N. R., & Sussman, S. (2013). Adolescent neurocognitive development, self-regulation, and school-based drug use prevention. *Prevention Science*, *14*(3), 218–228.

Posner, J., Polanczyk, G. V., & Sonuga-Barke, E. (2020). Attention-deficit hyperactivity disorder. *Seminar*, *395*(10222), 450–462.

Prehn-Kristensen, A., Molzow, I., Forster, A., Siebenhuhner, N., Gesch, M., Wiesner, C. D., & Baving, L. (2017). Memory consolidation of socially relevant stimuli during sleep in healthy children and children with attention-deficit/hyperactivity disorder and oppositional defiant disorder: What you can see in their eyes. *Biological Psychology*, *123*, 196–204.

Prehn-Kristensen, A., Molzow, I., Munz, M., Wilhelm, I., Muller, K., Freytag, D., Wiesner, C. D., & Baving, L. (2011). Sleep restores daytime deficits in procedural memory in children with attention-deficit/hyperactivity disorder. *Research in Developmental Disabilities*, *32*(6), 2480–2488.

Příhodová, I., Paclt, I., Kemlink, D., & Nevšímalová, S. (2012). Sleep microstructure is not altered in children with attention-deficit/hyperactivity disorder (ADHD). *Physiological Research*, *61*, 125–133.

Prihodova, I., Paclt, I., Kemlink, D., Skibova, J., Ptacek, R., & Nevsimalova, S. (2010). Sleep disorders and daytime sleepiness in children with attention-deficit/hyperactivity disorder: A two-night polysomnographic study with a multiple sleep latency test. *Sleep Medicine*, *11*, 922–928.

Punja, S., Shamseer, L., Hartling, L., Urichuk, L., Vandermeer, B., Nikles, J., & Vohra, S. (2016). Amphetamines for attention deficit hyperactivity disorder (ADHD) in children and adolescents. *Cochrane Database of Systematic Reviews*, *2*, CD009996.

Raman, S., & Coogan, A. N. (2019). Closing the loop between circadian rhythms, sleep, and attention deficit hyperactivity disorder. In H. C. Dringenberg (Ed.), *Handbook of behavioral neuroscience* (Vol. 30, pp. 707–716). Elsevier.

Reynaud, E., Forhan, A., Heude, B., Charles, M. A., & Plancoulaine, S. (2018). Association of night-waking and inattention/hyperactivity symptoms trajectories in preschool-aged children. *Science Report*, *8*, 15412.

Ricketts, E. J., Sturm, A., McMakin, D. L., McGuire, J. F., Tan, P. Z., Smalberg, F. B., McCracken, J. T., Colwell, C. S., & Piacentini, J. (2018). Changes in sleep problems across attention-deficit/hyperactivity disorder treatment: Findings from the multimodal treatment of attention-deficit/hyperactivity disorder study. *Journal of Child and Adolescent Psychopharmacology*, *28*(10), 690–698.

Ringli, M., Souissi, S., Kurth, S., Brandeis, D., Jenni, O. G., & Huber, R. (2013). Topography of sleep slow wave activity in children with attention-deficit/hyperactivity disorder. *Cortex*, *49*(1), 340–347.

Rodopman-Arman, A., Perdahli-Fiş, N., Ekinci, O., & Berkem, M. (2011). Sleep habits, parasomnias and associated behaviors in school children with attention deficit hyperactivity disorder (ADHD). *Turkish Journal of Pediatrics*, *53*(04), 397–403.

Roenneberg, T., Kuehnle, T., Juda, M., Kantermann, T., Allebrandt, K., Gordijn, M., & Merrow, M. (2007). Epidemiology of the human circadian clock. *Sleep Medicine Reviews*, *11*(6), 429–438.

Rugino, T. A. (2018). Effect on primary sleep disorders when children with ADHD are administered guanfacine extended release. *Journal of Attention Disorders*, *22*(1), 14–24.

Sacco, L. B., Leineweber, C., & Platts, L. G. (2018). Informal care and sleep disturbance among caregivers in paid work: Longitudinal analyses from a large community-based Swedish cohort study. *Sleep*, *41*(2), zsx198.

Sadeh, A., Pergamin, L., & Bar-Haim, Y. (2006). Sleep in children with attention-deficit hyperactivity disorder: A meta-analysis of polysomnographic studies. *Sleep Medicine Reviews*, *10*(6), 381–398.

Sadeh, A., Raviv, A., & Gruber, R. (2000). Sleep patterns and sleep disruptions in school-age children. *Developmental Psychology*, *36*(3), 291.

Saito, Y., Kaga, Y., Nakagawa, E., Okubo, M., Kohashi, K., Omori, M., Fukuda, A., & Inagaki, M. (2019). Association of inattention with slow-spindle density in sleep EEG of children with attention deficithyperactivity disorder. *Brain and Development*, *41*(9), 751–759.

Saletin, J. M., Coon, W. G., & Carskadon, M. A. (2017). Stage 2 sleep EEG sigma activity and motor learning in childhood ADHD: A pilot study. *Journal of Clinical Child & Adolescent Psychology*, *46*(2), 188–197.

San Mauro Martín, I., Blumenfeld Olivares, J. A., Garicano Vilar, E., Echeverry López, M., García Bernat, M., Quevedo Santos, Y., Blanco López, M., Elortegui Pascual, P., Borregon Rivilla, E., & Rincón Barrado, M. (2018). Nutritional and environmental factors in attention-deficit hyperactivity disorder (ADHD): A cross-sectional study. *Nutritional Neuroscience*, *21*(9), 641–647.

Santhi, N., Lazar, A. S., McCabe, P. J., Lo, J. C., Groeger, J. A., & Dijk, D. J. (2016). Sex differences in the circadian regulation of sleep and waking cognition in humans. *Proceedings of the National Academy of Sciences*, *113*(19), E2730–E2739.

Scammell, T. E., Arrigoni, E., & Lipton, J. O. (2017). Neural circuitry of wakefulness and sleep. *Neuron*, *93*(4), 747–765.

Scarpelli, S., Gorgoni, M., D'Atri, A., Reda, F., & De Gennaro, L. (2019). Advances in understanding the relationship between sleep and attention deficit-hyperactivity disorder (ADHD). *Journal of Clinical Medicine*, *8*(10), 1737.

Schmid, G., & Wolke, D. (2014). Preschool regulatory problems and attention-deficit/hyperactivity and cognitive deficits at school age in children born at risk: Different phenotypes of dysregulation? *Early Human Development*, *90*(8), 399–405.

Schwichtenberg, A. J., Christ, S., Abel, E., & Poehlmann-Tynan, J. A. (2016). Circadian sleep patterns in toddlers born preterm: Longitudinal associations with developmental and health concerns. *Journal of Developmental and Behavioral Pediatric*, *37*(5), 358.

Sciberras, E., Bisset, M., Hazell, P., Nicholson, J. M., Anderson, V., Lycett, K., Jongeling, B., & Efron, D. (2016). Health-related impairments in young children with ADHD: A community-based study. *Child: Care, Health and Development*, *42*(5), 709–717.

Sciberras, E., DePetro, A., Mensah, F., & Hiscock, H. (2015). Association between sleep and working memory in children with ADHD: A cross-sectional study. *Sleep Medicine*, *16*, 1192–1197.

Sciberras, E., Song, J. C., Mulraney, M., Schuster, T., & Hiscock, H. (2017). Sleep problems in children with attention-deficit hyperactivity disorder: Associations with parenting style and sleep hygiene. *European Child & Adolescent Psychiatry*, *26*(9), 1129–1139.

Scott, N., Blair, P. S., Emond, A. M., Fleming, P. J., Humphreys, J. S., Henderson, J., & Gringras, P. (2013). Sleep patterns in children with ADHD: A population-based cohort study from birth to 11 years. *Journal of Sleep Research*, *22*(2), 121–128.

Shrivastava, D., Jung, S., Saadat, M., Sirohi, R., & Crewson, K. (2014). How to interpret the results of a sleep study. *Journal of Community Hospital Internal Medicine Perspectives*, *4*(5), 24983.

Silvestri, R., Gagliano, A., Arico, I., Calarese, T., Cedro, C., & Bruni, O. (2009). Sleep disorders in children with attention-deficit/hyperactivity disorder (ADHD) recorded overnight by video-polysomnography. *Sleep Medicine*, *10*, 1132–1138.

Slama, H., Fery, P., Verheulpen, D., Vanzeveren, N., & Van Bogaert, P. (2015). Cognitive improvement of attention and inhibition in the late afternoon in children with attention-deficit hyperactivity disorder (ADHD) treated with osmotic-release oral system methylphenidate. *Journal of Child Neurology*, *30*(8), 1000–1009.

Smith, M. T., McCrae, C. S., Cheung, J., Martin, J. L., Harrod, C. G., Heald, J. L., & Carden, K. A. (2018). Use of actigraphy for the evaluation of sleep disorders and circadian rhythm sleep-wake disorders: An American Academy of Sleep Medicine systematic review, meta-analysis, and GRADE assessment. *Journal of Clinical Sleep Medicine*, *14*(7), 1209–1230.

Soehner, A. M., Bertocci, M. A., Levenson, J. C., Goldstein, T. R., Rooks, B., Merranko, J., Hafeman, D., Diler, R., Axelson, D., Goldstein, B. I., Hickey, M. B., Monk, K., Phillips, M. L., & Birmaher, B. (2019). Longitudinal associations between sleep patterns and psychiatric symptom severity in high-risk and community comparison youth. *Journal of the American Academy of Child & Adolescent Psychiatry*, *58*(6), 608–617.

Sonne, T., Müller, J., Marshall, P., Obel, C., & Grønbæk, K. (2016). Changing family practices with assistive technology: MOBERO improves morning and bedtime routines for children with ADHD. In *Proceedings of the 2016 CHI conference on human factors in computing systems*, May 07−12, 2016, Santa Clara, CA, USA.

Sonuga-Barke, E. J., Coghill, D., Wigal, T., DeBacker, M., & Swanson, J. (2009). Adverse reactions to methylphenidate treatment for attention-deficit/hyperactivity disorder: Structure and associations with clinical characteristics and symptom control. *Journal of Child and Adolescent Psychopharmacology*, *19*(6), 683–690.

Storebø, O. J., Pedersen, N., Ramstad, E., Kielsholm, M. L., Nielsen, S. S., Krogh, H. B., Moreira-Maia, C. R., Magnusson, F. L., Holmskov, M., Gerner, T., Skoog, M., Rosendal, S., Groth, C., Gillies, D., Buch Rasmussen, K., Gauci, D., Zwi, M., Kirubakaran, R., Håkonsen, S. J., . . . Gluud, C. (2018). Methylphenidate for attention deficit hyperactivity disorder (ADHD) in children and adolescents–Assessment of adverse events in non-randomised studies. *Cochrane Database of Systematic Reviews*, *5*(5), CD012069.

Storebø, O. J., Simonsen, E., & Gluud, C. (2016). Methylphenidate for attention-deficit/hyperactivity disorder in children and adolescents. *JAMA*, *315*(18), 2009–2010.

Suratt, P. M., Diamond, R., Barth, J. T., Nikova, M., & Rembold, C. (2011). Movements during sleep correlate with impaired attention and verbal and memory skills in children with adenotonsillar hypertrophy suspected of having obstructive sleep disordered breathing. *Sleep Medicine*, *12*(4), 322–328.

Tarakçioğlu, M. C., Kadak, M. T., Gürbüz, G. A., Poyraz, B. Ç., Erdoğan, F., & Aksoy, U. M. (2018). Evaluation of the relationship between attention deficit hyperactivity disorder symptoms and chronotype. *Archives of Neuropsychiatry*, *55*(1), 54.

Thomas, S., Lycett, K., Papadopoulos, N., Sciberras, E., & Rinehart, N. (2018). Exploring behavioral sleep problems in children with ADHD and comorbid autism spectrum disorder. *Journal of Attention Disorders*, *22*(10), 947–958.

Tong, L., Xiong, X., & Tan, H. (2016). Attention-deficit/hyperactivity disorder and lifestyle-related behaviors in children. *PLoS One*, *11*(9), e0163434.

Tong, L., Ye, Y., & Yan, Q. (2018). The moderating roles of bedtime activities and anxiety/depression in the relationship between attention-deficit/hyperactivity disorder symptoms and sleep problems in children. *BMC Psychiatry*, *18*(1), 298.

Touchette, E., Côté, S. M., Petit, D., Liu, X., Boivin, M., Falissard, B., Tremblay, R. E., & Montplaisir, J. Y. (2009). Short nighttime sleep-duration and hyperactivity trajectories in early childhood. *Pediatrics*, *124*(5), e985–e993.

Tsai, M. H., Hsu, J. F., & Huang, Y. S. (2016). Sleep problems in children with attention deficit/hyperactivity disorder: Current status of knowledge and appropriate management. *Current Psychiatry Reports*, *18*(8), 76.

Tsai, F. J., Tseng, W. L., Yang, L. K., & Gau, S. S. (2019). Psychiatric comorbid patterns in adults with attention-deficit hyperactivity disorder: Treatment effect and subtypes. *PLoS One*, *14*(2), e0211873.

Tso, W., Chan, M., Ho, F. K., Rao, N., Li, A. M., Chan, K. L., Tiwari, A., Wong, I. C. K., Wing, Y. K., Van Voorhees, B., Li, S. L., Goh, W. H. S., & Ip, P. (2019). Early sleep deprivation and attention-deficit/hyperactivity disorder. *Pediatric Research*, *85*(4), 449–455.

Um, Y. H., Jeong, J. H., Hong, S. C., Kim, T. W., Lim, H. K., Seo, H. J., & Han, J. H. (2016). Association between sleep parameters and cognitive function in drug-naïve children with attention-deficit hyperactivity disorder: A polysomnographic study. *Sleep Medicine*, *21*, 165–170.

Van der Heijden, K. B., Stoffelsen, R. J., Popma, A., & Swaab, H. (2018). Sleep, chronotype, and sleep hygiene in children with attention-deficit/hyperactivity disorder, autism spectrum disorder, and controls. *European Child & Adolescent Psychiatry*, *27*(1), 99–111.

Visser, S. N., Bitsko, R. H., Danielson, M. L., Perou, R., & Blumberg, S. J. (2010). Increasing prevalence of parent-reported attention-deficit/hyperactivity disorder among children—United States, 2003 and 2007. *Morbidity and Mortality Weekly Report*, *59*(44), 1439–1443.

Vitaterna, M. H., Takahashi, J. S., & Turek, F. W. (2001). Overview of circadian rhythms. *Alcohol Research & Health*, *25*(2), 85.

Wajszilber, D., Santiseban, J. A., & Gruber, R. (2018). Sleep disorders in patients with ADHD: impact and management challenges. *Nature and Science of Sleep*, *10*, 453.

Waldon, J., Vriend, J., Davidson, F., & Corkum, P. (2018). Sleep and attention in children with ADHD and typically developing peers. *Journal of Attention Disorders*, *22*(10), 933–941.

Walters, A. S., Silvestri, R., Zucconi, M., Chandrashekariah, R., & Konofal, E. (2008). Review of the possible relationship and hypothetical links between attention deficit hyperactivity disorder (ADHD) and the simple sleep related movement disorders, parasomnias, hypersomnias, and circadian rhythm disorders. *Journal of Clinical Sleep Medicine*, *4*(6), 591–600.

Wang, L. J., Huang, Y. S., Hsiao, C. C., & Chen, C. K. (2017). The trend in morning levels of salivary cortisol in children with ADHD during 6 months of methylphenidate treatment. *Journal of Attentional Disorders*, *21*(3), 254–261.

Wehmeier, P. M., Schacht, A., Wolff, C., Otto, W. R., Dittmann, R. W., & Banaschewski, T. (2011). Neuropsychological outcomes across the day in children with attention-deficit/hyperactivity disorder treated with atomoxetine: Results from a placebo-controlled study using a computer-based continuous performance test combined with an infra-red motion-tracking device. *Journal of Child and Adolescent Psychopharmacology*, *21*(5), 433–444.

Weiss, M. D., Wasdell, M. B., Bomben, M. M., Rea, K. J., & Freeman, R. D. (2006). Sleep hygiene and melatonin treatment for children and adolescents with ADHD and initial insomnia. *Journal of the American Academy of Child & Adolescent Psychiatry*, *45*(5), 512–519.

Wiebe, S., Carrier, J., Frenette, S., & Gruber, R. (2013). Sleep and sleepiness in children with attention deficit/hyperactivity disorder and controls. *Journal of Sleep Research*, *22*(1), 41–49.

Wiesner, C. D., Molzow, I., Prehn-Kristensen, A., & Baving, L. (2017). Sleep-dependent consolidation of rewarded behavior is diminished in children with attention deficit hyperactivity disorder and a comorbid disorder of social behavior. *Frontiers in Psychology*, *8*, 167.

Wigal, S. B., Kollins, S. H., Childress, A. C., Squires, L., & 311 Study Group. (2009). A 13-hour laboratory school study of lisdexamfetamine dimesylate in school-aged children with attentiondeficit/hyperactivity disorder. *Child and Adolescent Psychiatry and Mental Health*, *3*(1), 17.

Wigal, S. B., Wong, A. A., Jun, A., Stehli, A., Steinberg-Epstein, R., & Lerner, M. A. (2012). Adverse events in medication treatment-naive children with attention-deficit/hyperactivity disorder: Results from a small, controlled trial of lisdexamfetamine dimesylate. *Journal of Child and Adolescent Psychopharmacology*, *22*(2), 149–156.

Williams, K. E., & Sciberras, E. (2016). Sleep and self-regulation from birth to 7 years: A retrospective study of children with and without attention-deficit hyperactivity disorder at 8 to 9 years. *Journal of Developmental Behavioral Paediatrics*, *37*, 385–394.

Yazdanbakhsh, K., Aivazy, S., & Moradi, A. (2018). The effectiveness of response inhibition cognitive rehabilitation in improving the quality of sleep and behavioral symptoms of children with attention-deficit/hyperactivity disorder. *Journal of Kermanshah University of Medical Sciences*, *22*, e77114.

Yehuda, S., Rabinovitz-Shenkar, S., & Carasso, R. L. (2011). Effects of essential fatty acids in iron deficient and sleep-disturbed attention deficit hyperactivity disorder (ADHD) children. *European Journal of Clinical Nutrition*, *65*(10), 1167–1169.

Young, J., Rugino, T., Dammerman, R., Lyne, A., & Newcorn, J. H. (2014). Efficacy of guanfacine extended release assessed during the morning, afternoon, and evening using a modified

conners' parent rating scale–revised: Short form. *Journal of Child and Adolescent Psychopharmacology*, *24*(8), 435–441.

Yürümez, E., & Kılıç, B. G. (2016). Relationship between sleep problems and quality of life in children with ADHD. *Journal of Attention Disorders*, *20*(1), 34–40.

Zambrano-Sánchez, E., Martínez-Cortés, J. A., Dehesa-Moreno, M., del Río-Carlos, Y., & Poblano, A. (2013). Correlation between sleep disorder screening and executive dysfunction in children with attention deficit-hyperactivity disorder. *Arq Neuropsiquiatr*, *71*, 896–901.

Zarinara, A. R., Mohammadi, M. R., Hazrati, N., Tabrizi, M., Rezazadeh, S. A., Rezaie, F., & Akhondzadeh, S. (2010). Venlafaxine versus methylphenidate in pediatric outpatients with attention deficit hyperactivity disorder: A randomized, double blind comparison trial. *Human Psychopharmacology: Clinical and Experimental*, *25*(7–8), 530–535.

Author Biographies

Upasana Bondopadhyay completed her BA in Psychology from Delhi University India and M.Phil. in Clinical Psychology from Amity University, India. She is a clinical psychologist by training registered with the Health and Care Professions Council, UK, and the Rehabilitation Council of India. Upasana is currently pursuing PhD in Psychology from National University of Ireland, Maynooth. Her research interests include childhood neurodevelopmental disorders, child and adolescent psychotherapeutic interventions and psycho-diagnostic testing.

Unai Díaz-Orueta, PhD, received his doctorate in psychology from the University of Deusto, Spain in 2006. Since 2000, he has worked as a clinical psychologist in a variety of settings and got the validation to work as a Clinical Psychologist in Ireland by the Department of Health and Children in 2007. From 2012 to 2015, he worked as a researcher and then as the R+D+I Director of Nesplora SL developing virtual reality based neuropsychological assessment and psychological intervention tools. He was been a Marie Sklodowska-Curie Actions Individual Fellow (Research Fellow) at Dublin City University (Dublin, Ireland) working on the EC funded project "E-SPACE: European Standardised Process Approach to Cognitive Evaluation in older people" (2016-2018) (http://www.e-spacedementiaproject.eu). He is currently Lecturer/Assistant Professor in Neuropsychology within the Department of Psychology at Maynooth University, Ireland.

Andrew N Coogan, PhD, received a doctorate from University College Dublin in 2000 for work on the neuroimmune modulation of synaptic plasticity in the hippocampus. Following this, he moved to work in the field of circadian rhythms and sleep, and he has headed a research group in this area since 2006. He has particular interests in the role of circadian clocks in attention and attention disorders in both children and adults. He is currently a professor and head of the Department of Psychology at Maynooth University, Ireland.